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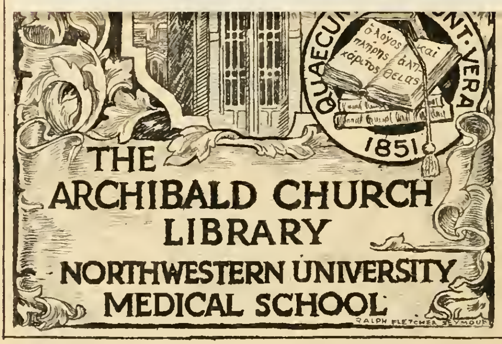
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INVESTIGATIONS

INTO THE

ETIOLOGY

OF

TRAUMATIC INFECTIVE DISEASES,

BY

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TRANSLATOR'S PREFACE.

The reader of the following work cannot fail to admit the beauty and importance of the observations which it records, provided he can be satisfied of their authenticity. With regard to the plates which illustrate the text I am able to give the most satisfactory confirmatory evidence. Since the work was published Dr. Koch has succeeded in photographing his microscopic preparations, and he has forwarded a considerable number of the photographs to Professor Lister. These, which have unmistakably been taken from sections of tissue, when examined by a pocket lens or projected on a screen, show plainly that the drawings are faithful representations of what has been seen. I have also lately received from Dr. Koch several of his stained sections, and those which I have been able to examine are equally satisfactory with the photographs.

PREFACE.

THE present work pertains to a series of investigations which I have already made regarding the etiology of infective diseases, and which I intend to prosecute more fully hereafter. The object of the enquiry was to determine whether the infective diseases of wounds are of parasitic origin or not. Owing to extraneous circumstances I found it necessary to confine myself solely to experiments on the action of putrid materials on animals. These experiments have led to definite, and, as it seems to me, not unimportant results. Nevertheless, in order to obtain a complete answer to the question it would have been necessary to carry out a further series of similar experiments on animals with materials obtained from persons suffering from or who had died of traumatic infective diseases, and—what indeed seems to me to be the most important—to look for micro-organisms in the human body by the method described in this work.

As, however, I had not the opportunity of completing my investigations in this direction, I have contented myself with producing experimentally in animals morbid processes which resemble the traumatic infective diseases observed in man, and which may serve as illustrations of them.

Since I have succeeded in completing this series of morbid processes, to the extent of furnishing examples illustrative of the most important traumatic infective diseases, *viz.*, of septicæmia, pyæmia, progressive suppuration, gangrene, and erysipelas, I believe that I have solved the problem, so

far as is possible by experiments on animals alone; and I therefore think it well to publish the results already obtained.

With respect to the illustrations accompanying this work I must here make a remark. In a former paper* on the examination and photographing of bacteria I expressed the wish that observers would photograph pathogenic bacteria, in order that their representations of them might be as true to nature as possible. I thus felt bound to photograph the bacteria discovered in the animal tissues in traumatic infective diseases, and I have not spared trouble in the attempt. The smallest, and, in fact, the most interesting bacteria, however, can only be made visible in animal tissues by staining them, and by thus gaining the advantage of colour. But in this case the photographer has to deal with the same difficulties as are experienced in photographing coloured objects, *e. g.*, coloured tapestry. These have, as is well known, been overcome by the use of coloured collodion. This led me to use the same method for photographing stained bacteria, and I have, in fact, succeeded, by the use of eosin-collodion, and by shutting off portions of the spectrum by coloured glasses, in obtaining photographs of bacteria which had been stained with blue and red aniline dyes. Nevertheless, from the long exposure required and the unavoidable vibrations of the apparatus, the picture does not have sharpness of outline sufficient to enable it to be of use as a substitute for a drawing, or indeed even as evidence of what one sees. For the present, therefore, I must abstain from publishing photographic representations; but I hope, at a subsequent period, when improved methods allow a shorter exposure, to be able to remedy this defect.

* The paper referred to is published in Cohn's Beiträge zur Biologie d. Pflanzen.

INTRODUCTION.

As at present used, the term "traumatic infective diseases" (Wund infections Krankheiten) indicates a group of affections formerly known as traumatic fever, purulent infection, putrid infection, septicæmia, pyæmia, but which were included at a subsequent period (when the view became generally accepted that these diseases were essentially of the same nature) under the title "pyæmic or septicæmic processes."

Strictly speaking, we should include in this group all those diseases which are the sequel of wounds, even the smallest, as, for example, the pricks of a vaccinating needle, and which have been proved with certainty, by clinical observation or experiment, to be contagious. For example, vaccine infection, anthrax, glanders, hydrophobia, and even syphilis, must be ranked among the traumatic infective diseases. Nevertheless this term does not commonly have such a wide signification, but is limited to those morbid processes specially interesting to the surgeon, which may complicate injuries and operation wounds; in other words, to septicæmia, pyæmia, progressive inflammation and suppuration, and erysipelas. Of late the conviction has more and more gained ground that puerperal fever is also to be regarded as an infective disease, starting from the placental surface or from wounds of the genital passages. Further, many authors include diphtheria also among these diseases, because it at times attacks wounds, and because the possibility of transmitting it by inoculation has been abundantly demonstrated.

In the comments introductory to the experimental part of this work, I shall likewise confine myself to the last-mentioned morbid processes; in the second part, however, I shall deviate from the ordinary limitation of the term "traumatic infective diseases" in so far that I shall also take notice of anthrax on account of its manifold relations to the septicæmia produced experimentally in animals.

The expressions pyæmia and septicæmia are often used with different meanings, and it is therefore necessary to indicate precisely what I shall understand by terms so universally employed.

For a long time pyæmia was distinguished from septicæmia by the occurrence of metastatic deposits in the former and their absence in the latter. But since it has been established that, even in such cases as had been previously described as septicæmia, isolated microscopic metastatic deposits are not unfrequently present, and that the two processes cannot be definitely separated in this way, some authors have preferred to designate as septicæmia the disease brought about by absorption of dissolved putrid poison, and to call all the other morbid processes connected with the development of microscopic organisms, pyæmic processes.

Birch-Hirschfeld,* for example, separates pyæmia from septicæmia in this way. He understands by the term "septicæmia" a disease originating in alterations of the blood, which alterations are a consequence of the absorption of the products of putrefaction. On the other hand, he defines pyæmia as "a general infection, which proceeds from the surfaces of wounds or from the focus of a primary suppurative inflammation, probably evoked by specific organisms and different from the putrid infection." Cohnheim† also identifies

* Lehrbuch der pathologischen Anatomie. Leipzig, Vogel, 876, p. 1224.

† Vorlesungen uber Pathologie. Berlin, 1877, s. 469.

septicæmia with putrid infection, and attributes it to the entrance of a putrid poison in solution into the fluids of the body. Davaine, whose works I shall have to allude to repeatedly, adheres, on the other hand, to the older distinction between pyæmia and septicæmia, and includes under the latter term all those cases in which the post-mortem examination shows no metastatic deposits, although in both cases he considers the co-operation of specific organisms as proven.

The terms pyæmia and septicæmia no longer retain their original signification, for pyæmia does not arise, as was at one time believed, from the entrance of pus into the blood-vessels, and septicæmia is not putrefaction of the living blood. They now remain only as collective names for a number of symptoms which in all probability belong to different diseases. So long, however, as these diseases are not sufficiently separated from each other, it seems best for the present to retain these terms in their ordinary signification, in order to avoid the necessity of constantly adopting new definitions.

For these reasons I shall in what follows include under the term septicæmia all those cases of general traumatic infection in which no metastatic deposits occur, and under pyæmia those in the course of which they may be present.

THE

PRESENT STATE OF KNOWLEDGE

WITH REGARD TO

THE RELATIONS OF MICRO-ORGANISMS TO TRAUMATIC INFECTIVE DISEASES.

I. The Occurrence of Micro-organisms in the Human Body in Disease.

The first communication regarding the occurrence of bacteria in the organs of those who have died of traumatic infective diseases was made by Rindfleisch* in the year 1866. In pyæmia, puerperal fever, and like infective diseases, small softened metastatic deposits, of the size of a pin's head, at times occur. These deposits are always found in greater numbers in the substance of the heart, in the muscular tissue of which they have at first the appearance of greyish white spots, these specks becoming, at a later period, cavities filled with a thin fluid pulp. The contents of these cavities consist, as Rindfleisch has shown, not of pus-corpuscles, but solely of "vibriones." These organisms lie at first closely packed between the fasciculi of the muscle; at a later period, however, from disintegration of the muscular fibres, they penetrate into their interior. Rindfleisch was unable to trace the alterations further than to the formation of these small abscess-like softened spots, because the process in question only occurs in those forms of infective diseases which are most severe and rapidly fatal. Rindfleisch has not specially described the organisms termed by him "vibriones," as regards their size or as to whether they were rod-shaped or spherical.

* Lehrbuch der pathologischen gewebelehre. 1 Aufl., s. 204 (4 Aufl., p. 199).

That the development of these miliary purulent deposits, which occur also in other organs in typhus, pyæmia, &c., is produced by parasitic organisms, in other words by bacteria, was shown almost simultaneously by Von Recklinghausen and Waldeyer. Von Recklinghausen* describes the bodies found in the smallest renal veins, in the glomeruli, urinary tubules, and pulmonary alveoli, under the name of micrococci, and states that they may be distinguished from detritus by the uniform size of their granules and by their resistance to the action of glycerine, acetic acid, caustic soda, &c. He likewise calls attention to the brownish colour of the centre of the deposit, as well as to the fact that the uriniferous tubules and the vessels in which the micrococci lie are very much distended at intervals.

Waldeyer confirmed Rindfleisch's statement regarding the occurrence of numerous miliary bacteric deposits in the substance of the heart in pyæmia, and he also found bacteria in small abscess-like spots in the kidneys.

Attention was directed by these investigations to the bacteria, which are present in metastatic deposits in pyæmia, and which up to that time had been overlooked or regarded as unimportant. These statements were confirmed and extended by many similar observations, and it may now be regarded as an established fact that, in most of the metastatic deposits in pyæmia, bacteria in the form of the so-called zooglæa will, on careful examination, be found. Nothing essentially new, with some exceptions to be noticed directly, has, however, been added by later investigations to the original observations of Rindfleisch, Von Recklinghausen, and Waldeyer. It is therefore unnecessary to take special notice of the numerous papers on this subject.

It is worthy of mention that P. Vogt† has seen moving "monads," even during life, in the metastatic deposits of a pyæmic individual. It now naturally occurred to observers to subject the pus of wounds to examination, in order to ascertain if the bacteria, found in the metastatic deposits, accumu-

* Vortrag in der Würzb. physik.-med. Ges. 10 Juni, 1871 (quoted from Birch-Hirschfeld, Med. Jahrbb. Bd. 155, Heft 1).

† Centralblatt für die medicin, Wissenschaft. 1872, No. 44.

lated in the first instance in the pus of the wound, and from thence penetrated into the tissue. Extensive observations of this kind have been made by Birch-Hirschfeld.* He came to the conclusion that the unhealthiness of a wound stood in a direct relation to the number of spherical bacteria in the pus of that wound. The more abundantly these appeared the worse became the state of the wound and the general condition of the patient. The most unfavourable cases were those in which the spherical bacteria had become grouped together in colonies (zooglæa). As the spherical bacteria increased in number their penetration into the pus-corpuscles could also be observed. Birch-Hirschfeld examined at the same time the blood of pyæmic patients, and found that it contained bacteria. He further states that the severity and rapidity of the general infection are in proportion to the number of bacteria which may be detected in the blood.

The channel by which the bacteria gain access to the metastatic deposits would, if these observations were correct, be pretty clearly indicated. The mode by which they pass into the circulation, from the surface of the wound, alone remained unknown. This blank was filled up by the investigations of Klebs. The work of Klebs† deserves notice here, not on this ground alone, but also because his researches furnish very numerous and thorough observations on the bacteria of wound diseases, and further because in them the attempt was made for the first time, by the aid of abundant and excellently used materials for observation, to demonstrate a causal connection between bacteria and traumatic infective diseases. Klebs designates the bacteria found in the pus of a wound as microsporon septicum, starting with the view that spherical and rod-shaped bacteria stand in a genetic relation to each other, and also that the micrococci and bacteria commonly found together in wounds are forms of the same organism. The growth of this microsporon septicum in the form of zooglæa masses firmly attached to the surface of the wound was observed by Klebs on granulations, joint surfaces, and serous membranes. He was also able to trace the penetration

* Untersuchungen über Pyæmie. Leipzig, 1873.

† Beiträge zur patholog. Anatomie der Schusswunden. Leipzig, Vogel, 1872.

of the zooglœa into the interspaces of the cellular tissue. This takes place either with or without the aid of wandering lymph corpuscles. The passage of the microsporon along the lymphatic vessels could not be followed with complete certainty; on the other hand its penetration through the eroded walls of a vein into the circulation was observed in one instance. Further, the elements of the microsporon were found by Klebs in the thrombi which develop behind the valves of veins, and in the metastatic deposits in the lung and liver.

Although the facts which have been hitherto collected with reference to the dependence of pyæmia on the development of bacteria in the body are numerous and important, yet the statements relating to the occurrence of organisms in *septicæmia* are few and doubtful.

Coze and Feltz as well as Hueter* attribute the corrugated form of the red corpuscles frequently seen in septicæmic diseases to the adhesion and penetration of bacteria; an observation which has been much and justly doubted.

The only other statement I have been able to find, as to the presence of bacteria in the blood in septicæmia, is made by Collmann von Schatteburg †. He saw, in one case, rods both in the blood of the body generally and in the vascular loops of the glomeruli.

The observations on erysipelas are much more abundant.

Nepveu ‡ found micrococci in the blood of erysipelatous patients, and these were present in greatest number in blood taken from the erysipelatous part.

Wilde § obtained the same result and he also states that the pus of wounds, from which erysipelatous inflammation starts, contains numerous micrococci. Orth ‖ has also found micrococci in the contents of the bullæ in erysipelas.

Of especial importance is the discovery made by Von Recklinghausen and Lukomsky¶ that the lymphatic vessels

* Compare Birch-Hirschfeld: Lehrbuch der pathologischen Anatomie. Leipzig, 1876, p. 469, and Med. Jahrbb., Bd. 166, p. 184.

† Virchow und Hirsch.: Jahresbericht for 1875. I., page 369.

‡ Virchow und Hirsch: Jahresbericht for 1872. I., page 254.

§ Med. Jahrbb. Bd. 155, Heft 1, page 104.

‖ Archiv f. Experiment. Pathol. u. Pharmakol. I., page 81.

¶ Virchow's Archiv. Bd. 60, page 418.

and canaliculi (saft kanälchen) of the skin at the border of the erysipelatous part are filled with micrococci.

This observation was confirmed by Billroth and Ehrlich,[*] who likewise found micrococci, not only in the lymphatic, but also in the blood-vessels.

Micrococci have also been seen, by Tillmanns,[†] in erysipelatous skin, and by Letzerich[‡] in cases of erysipelas attacking vaccination wounds, in the wound itself, in the blood-vessels, muscles, liver, spleen, and kidneys.

With respect to phlegmonous suppurations, the observations have apparently been confined as yet to the contents of the abscesses, while the walls of the latter—that is to say the adjacent tissue which, as will subsequently be shown, is the true seat of the bacteric development—have up to this time received no attention. In the pus from the abscess, just as in the ordinary pus from wounds, bacteria have often and micrococci have almost always been found. A detailed account of the statements on this point is accordingly unnecessary.

Hospital gangrene differs so little from the diphtheria of mucous membranes that the observations made with reference to the latter also hold good for the former.

According to Cohnheim,[§] after tracheotomy the disease at times spreads from the mucous membrane to the operation wound. But even without any apparent infection wounds often become diphtheritic, and Cohnheim thinks it probable that hospital gangrene is nothing more or less than a diphtheritic inflammation of the surface of a wound.

Hueter[||] found in the greyish diphtheritic deposits on wounds, and, on more accurate examination, in the neighbouring tissues, as yet apparently quite healthy, the same small round bodies with dark contours which he afterwards saw in the false membrane in diphtheritis of the larynx and pharynx.

* v. Langenbeck's Archiv. Bd. 20, p. 418.

† Deutsche medicin. Wochenschrift, 1878. No. 17, p. 224.

‡ Virchow und Hirsch: Jahresbericht for 1875, p. 69.

§ *L. c.*, p. 482.

|| Steudener: Volkmann's klinische Vorträge. No. 38, p. 24.

By the researches of Oertel, Nassiloff, Classen, Letzerich, Klebs, and Eberth,* it has been placed beyond doubt that in diphtheritic deposits large numbers of micrococci are present. The statements are, however, as yet contradictory with regard to the question whether or not the bacteria penetrate into the tissues.

Oertel † found the inflamed mucous membrane crammed with micrococci, and was further able to trace them in the afferent lymphatic vessels of the nearest lymphatic glands, in the glands themselves, as well as in the blood-vessels of the kidney and of other internal organs.

Similar observations have been made by Eberth, Nassiloff, and Letzerich.

The presence of small deposits of bacteria in the cardiac tissue, in the liver, kidneys, and other organs, in cases of diphtheria, has of late been repeatedly demonstrated.‡

Attention has also been drawn, by different observers, § especially by those who have inoculated diphtheria on the cornea of rabbits, to the brownish colour of the micrococcus masses.

On comparing the behaviour of the bacteria in diphtheria and in pyæmia, one is at once struck by a remarkable correspondence. In both morbid processes the surface of the wound is covered with masses of micrococci which penetrate into the deeper layers of the tissue and into the lymphatic vessels; in both, peculiar miliary bacteric deposits are present in the cardiac tissue, in the liver, and in the kidney; and in both, these bacteric deposits are of a brownish colour. The question is at once forced on the mind, May not the parasitic micro-organisms of pyæmia and of diphtheria be identical?

The same appearances may be seen in puerperal fever. In this disease Waldeyer || has found spherical bacteria in the affected tissues, in the lymphatic vessels, and in the peritoneal exudation; whilst Birch-Hirschfeld¶ has observed

* *See* Birch-Hirschfeld: Lehrbuch der pathol. Anatomie, p. 799.

† Steudener, *l. c.*, p. 24.

‡ Cohnheim, *l. c.*, page 480.

§ Birch-Hirschfeld, *l. c.*, page 799.

|| Archiv für Gynäkologie. Bd. II. 1871.

¶ Med. Jahrbb. Bd. 155, p. 105.

micrococcus masses on vaginal ulcers, in the perivaginal cellular tissue, in the blood, in the spleen, and in the liver. The presence of micrococci in the kidneys, lungs, and cardiac muscular tissue was demonstrated by Heiberg and Orth,* and the latter makes mention of the greyish yellow colour of those present in the uriniferous tubules which are affected with nodular dilatations.

As standing probably in close relation to puerperal fever, we must here mention the disease affecting new-born infants, first described by Orth † and called by him *mycosis septica*. In one such case micrococci were found in the blood, in the pleural cavity, and in the urinary bladder.

The so-called mycosis of the navel in new-born infants (nabelmykose) seems likewise to belong to this group of diseases. Weigert ‡ describes a case of this kind, and states that the ulcer of the navel was covered with micrococci, and that groups of micrococci were present in the centre of small extravasations of blood in the lungs and kidneys.

Hennig has investigated an analogous case and obtained the same result.

The extremely interesting observations with regard to the occurrence of bacteria in endocarditis seem less easily explicable.

All investigators who have been engaged in seeking bacteria in morbid tissues agree in regarding the undertaking as one of extraordinary, often even of insuperable, difficulty. To make up for the weakness of the anatomical proofs as to the presence of bacteria, pathological experiment has in most cases been resorted to. In order, therefore, to obtain a complete survey of the facts known respecting the relations of bacteria to traumatic infective diseases, it is now necessary to give a short digest of the results of the experimental investigations on this subject.

* *Ibid.* Bd. 166, p. 188.

† Archiv. der Heilkunde, 1872. XIII., p. 265.

‡ Jahresber. der schles. Gesellsch. für vaterl. Kultur. 1875, p. 229.

II. Relations of Micro-organisms to Traumatic Infective Diseases, as established by Experiment.

It is known, as the result of experience, that, when traumatic infective diseases set in, the discharges of the wound and the fluids in the neighbouring tissues take on a putrid character. These alterations in the wound often, indeed, make their appearance before any perceptible manifestation of the general disease, and it was therefore concluded that the putrefaction of the discharges was the cause of the infective disease. Some, however, disputed the accuracy of this conclusion, and maintained that the infective disease was produced by causes acting from within, and that the deterioration of the wound occurred secondarily. To settle this controversy numerous experiments have been set on foot. Experimenters for a long time contented themselves with ascertaining the noxious influence of putrid substances on animals when injected into the blood or into the subcutaneous tissue, and with isolating the poisonous substance contained in these putrid fluids. The question as to whether the disease produced by the injection of the putrid fluid was only a simple poisoning, or whether it in reality possessed the infective qualities of those diseases observed in man, was left untouched by the older and most of the later experimenters. If in an animal, by injection of a putrid fluid, a disease was produced resembling to some extent the human infective disease in symptoms and post-mortem appearances, this circumstance sufficed for their identification, and from such an experiment extensive conclusions as to infective diseases were drawn. But in order that such experiments should prove the infective character of the disease, it must be definitely ascertained, by further transmission from one animal to another, that the disease produced experimentally is in like manner of undoubtedly infective nature.

As we have here to do only with infective diseases, all the investigations which have reference merely to the toxic properties of putrid materials, as well as those in which the possibility of a confusion between toxic action and infection is not excluded, must be left unnoticed.

The first attempt to produce traumatic infective diseases artificially in the lower animals was made by Coze and Feltz.* These investigators injected some grammes of blood, from a patient who had died of putrid poisoning and puerperal fever, into the subcutaneous cellular tissue of rabbits. In consequence of this the animals died with peculiar and characteristic symptoms. A much smaller quantity of the blood of the rabbits thus killed was injected subcutaneously into other rabbits and the same symptoms and fatal termination occurred as took place with the original putrid blood. Coze and Feltz continued this transmission of blood, in gradually diminishing quantities, from the dead animals to others, and they finally succeeded in bringing about infection with an extremely minute amount of blood. This led them to assume that the poison increased in virulence by successive inoculations. In the blood of animals which had died of putrid infection they found bacteria in great numbers, indeed they assert that they have seen at the same time rods, long threads with an oscillating or vermiform motion, and chains of small granules.

The discovery of the increasing virulence of the successively inoculated putrid poison excited the most lively interest.

The experiments of Coze and Feltz were repeated and confirmed by Clementi and Thin, and by Behier and Lionville.† These observers likewise convinced themselves that for the first infection a comparatively large quantity of the infective material, be it blood, peritoneal fluid, or the like, is necessary; while for the later infections an extremely minute quantity is sufficient. They also found numerous bacteria in the blood of the animals killed by the inoculation.

Colin, Vulpian, Raynaud, and others obtained similar results.‡

Davaine,§ however, has studied these conditions more thoroughly than any other observer. He transmitted the

* Virchow und Hirsch: Jahresbericht für 1866. I., p. 195.

† Richter: Die neueren Kenntnisse der Krankmachenden Schmarotzerpilze. separatabdr. aus d. med. Jahrbb.

‡ Med. Jahrbb. Vol. 166, p. 174.

§ *Ibid.*

infection through a series of twenty-five animals in succession, and for the last effective transmission of the putrid infecting material he used only a trillionth part of a drop of blood. Davaine saw in the blood of these animals actively moving bacteria, differing in that respect from those of splenic fever, which are quiescent and were for that reason called by him bacteridia.

Although the most diverse fluids were used by him in these experiments for the first infection, *e. g.*, putrefying blood, blood from pyæmic cases, from puerperal fever, scarlet fever, small-pox, and typhus, yet the effects produced were always similar, and the post-mortem examination showed in all cases bacteria in the blood, and swelling of the spleen, with absence of metastatic deposits. Davaine accordingly calls this disease septicæmia.

Of the other traumatic infective diseases, diphtheria and erysipelas have been artificially produced in animals.

The attempt to transmit erysipelas was first made by Orth.* He injected under the skin of a rabbit the contents of an erysipelatous bulla in which were numerous spherical bacteria. There followed an inflammation entirely analogous to erysipelas in man, and, by the application of the œdematous fluid from the subcutaneous tissue of this animal to a second rabbit, the characteristic progressive inflammation was communicated to the latter. In the œdematous fluid in the subcutaneous cellular tissue, and in the affected parts of the skin of the animals suffering from artificial erysipelas, Orth demonstrated the presence of bacteria in large numbers.

Lukomsky † has also experimented on rabbits with erysipelatous fluid in order to produce an artificial erysipelas. He, however, obtained in the animals experimented on an extensive phlegmonous inflammation of the subcutaneous cellular tissue with implication of the skin. But in his cases also micrococci were present in the canaliculi (saftkanälen) of the areolar tissue and in the lymphatic vessels.

That diphtheria may be communicated to rabbits, and that micrococci appear in the artificial diphtheria similar

* Birch-Hirschfeld: Lehrb. d. patholog. Anat., p. 608.

† *Loc. cit.*

in characters to those found in diphtheria in man, has been demonstrated by Hueter, Tommasi, Œrtel, and Letzerich (*l. c.*)

The investigations on diphtheria led to the introduction of an experiment of extreme importance in the study of pathogenic bacteria, *viz.*, the use of the transparent cornea of the rabbit as the place for inoculation.

Nassiloff* and Eberth† were the first to carry out these corneal inoculations. At first diphtheritic substances were alone used, but it was soon found that the most diverse putrid materials, products of inflammation, and the like, could also be inoculated on the cornea with effect.

Similar experiments have likewise been carried out and in various ways modified by Leber, Stromeyer, Dolschenkow, Orth, and more especially by Frisch.‡

In a successful inoculation of this kind a peculiar stellate patch with conical processes—the so-called "mushroom" appearance (Pilzfigur)—is produced, the centre of which is the point of inoculation. The materials composing this patch are, in diphtheritic inoculations, dense masses of micrococci, which are of a yellow or greenish-brown colour like the micrococci of the diphtheritic and pyæmic deposits in the muscular tissue of the heart and in the kidneys. The "Pilzfigur" is also obtained by inoculation of putrid fluids, and here it consists of rod-shaped bacteria. Frisch further inoculated materials from splenic fever on the cornea of living rabbits, and observed the development of splendid "Pilzfiguren," which consisted only of Bacillus Anthracis.§

In all these experiments the inflammatory appearances in the cornea were exactly in proportion to the development and extent of the bacteria. Eberth found the association of the bacteria with the artificial diphtheria of the cornea so

* Virchow's Archiv. Vol. 50, p. 550.

† Eberth. Bacterische Mykosen. Leipzig, 1872.

‡ Experimentelle Studien über die Verbreitung der Fäulnissorganismen in den Geweben. Erlangen, 1874.

§ Die Milzbrand-bakterien und ihre Vegetation in der lebenden Hornhaut. Vienna, 1876.

‖ *L. c.*, p. 14.

constant that he distinctly says: Without the fungi (*i. e.*, bacteria) no diphtheria.

A special method, and one of much promise for demonstrating the origin of infective diseases from a contagium animatum, was followed by Klebs.* He introduced fluids and other substances, taken from patients who were suffering from or who had died of infective diseases, into thoroughly purified flasks containing cultivating fluids. After development of organisms had occurred in these fluids a small quantity was taken and put into a second vessel containing a similar liquid. With the fluid of the second flask a third was inoculated, and so on through a series sufficiently long to enable him to assume that only an excessively small part of the infective substance originally employed, or indeed none at all, could be present in the last cultivating liquid. The fluids, thus freed from the original infective material, were inoculated on animals. Klebs† has employed this method, which he terms fractional cultivation, more especially with substances from diphtheritic and septic processes, though also with material from various other diseases. The fluids obtained in this manner by fractional cultivation, when inoculated on animals, produced again septicæmia and diphtheria; and Klebs also found, both in the cultivating fluids and in the infected animals, the characteristic micrococci.

In a similar manner Orth‡ has grown in cultivating fluids bacteria from an erysipelatous bulla, and, by the injection of this fluid, has reproduced erysipelas in rabbits.

III. Objections to the conclusiveness of these Facts.

The facts put together in the two foregoing sections are undoubtedly of considerable significance. When taken along with theoretical considerations, and looked at in the light of

* Virchow und Hirsch. Jahresbericht, 1874. Vol. i., p. 359.

† Ueber die Umgestaltung der medicinischen Anschauungen in den letzten drei Jahrzehnten. Rede, gehalten in München bei der 50. Versammlung deutscher Naturforscher. Leipzig. Vogel. 1878.

‡ Birch-Hirschfeld, *l. c.*, p. 608.

the brilliant results of the antiseptic method of treatment, they furnish evidence sufficient to enable many to accept as proved the existence of living infective material, especially in traumatic infective diseases. On the other hand, various objections, of more or less weight, have been urged against this assumption; and a short discussion of these is requisite in order to form a judgment as to the significance of bacteria in traumatic infective diseases.

A considerable number of investigators have advanced the statement that the normal blood and tissues of man and of the lower animals always contain micro-organisms. From this some infer that these organisms are not the cause of the infective disease, but that an abnormal increase in their numbers follows the morbid process, because the fluids of the animal body, when altered by disease, present conditions very favourable for their development. We need not consider these views, which have as yet never been experimentally proved, but which are advanced on theoretical grounds alone. Were it, however, true that bacteria do occur in normal blood, and that the same bacteria—*e. g.*, micrococci—are found, though in unusual numbers, in organs altered by disease, then the possibility of proving that these micrococci were the cause of the disease would be rendered much more difficult, perhaps indeed quite hopeless. We must therefore enquire how far the assertion in question is correct.

Lostorfer, Nedsvetzki, and Béchamp* discovered small moving particles in normal human blood. Lostorfer calls these bodies micrococci, and asserts that he has traced their further development to sarcinæ. Nedsvetzki has given to these particles the name of hæmococci, and he considers them as identical with the bodies described by Béchamp. Béchamp has in numerous papers expressed his views respecting the bodies called by him microzymes. He found these bodies in almost all animal fluids, and, from experiments which he carried on in conjunction with Estor, he concludes that microzymes can, through their physiological activity, bring about the coagulation of the blood, and the lactic, acetic,

* Richter, *l. c.*, p. 12.

and alcoholic fermentations; that they are also active in the transformation of glycogen in the liver, in the development of the embryo in hens' eggs during hatching, and in all possible processes in the animal body. That Béchamp looks on his microzymes as intimately related to bacteria is apparent, because, according to him, the microzymes in the intestine below the iliocæcal valve change normally into bacteria; and at diseased spots of the small intestine—as, *e. g.*, where a tapeworm is attached—bacteria develop immediately from microzymes.

J. Lüders, Bettelheim, Richardson,* and, later, Kolaczek and Letzerich,† also believe that they have seen bacteria in normal human blood.

Tiegel‡ and Billroth§ have attempted in an indirect manner to demonstrate the existence of bacteria in normal animal tissues. They introduced, with certain precautions, fresh portions of muscle, liver, &c., into melted paraffin. The tissues thus enclosed in an air-tight case were, after the lapse of some time, examined to ascertain the presence or absence of bacteria. Numerous bacteria were found, and hence Billroth‖ concludes that in most of the tissues of the body, in greater proportion indeed in the blood, spores of bacteria capable of development are present.

Objections have been urged against the experiments of Billroth and Tiegel, to the effect that the enclosure in paraffin does not protect against the entrance of bacteria, because cracks and fissures form in the paraffin on cooling, and even afterwards, as every one must have observed who has embedded objects in paraffin for microscopical examination.

When normal blood was tested by Pasteur,¶ Burdon Sanderson,** and Klebs,†† as to its power of causing develop-

* Virchow and Hirsch. Jahresbericht for 1868. Vol. i., p. 205.

† Med. Jahrbb. Vol. 168, p. 68.

‡ Virchow and Hirsch. Jahresber., 1874. Vol. i., p. 119.

§ Coccobacteria Septica. Berlin, 1874, p. 58.

‖ *L. c.*, pp. 60 and 137.

¶ Virchow und Hirsch. Jahresberich., 1874. Vol. i., p. 119.

** Cohn. Beiträge zur Biologie der Pflanzen. Vol. i., part 2, p. 194.

†† Med. Jahrbb. Vol. 166, p. 196.

ment, by methods excluding all sources of error, negative results were obtained.

Opposed to the alleged direct observations of bacteria in normal blood are the statements of trustworthy microscopists such as Rindfleisch and Riess,* who distinctly assert that the normal blood is free from bacteria, but that on the other hand, as Riess more especially pointed out, it contains small round bodies more or less numerous, which are most probably *débris* arising from the disintegration of white blood corpuscles, and which, on account of their resemblance to micrococci, have been confounded with them.

According to my own experience the examination of blood, with the view of ascertaining the possible presence of bacteria, is excessively difficult, unless one makes use of the aids to be described afterwards, *viz.*, of staining and suitable illumination. Without the assistance derived from these methods it is in most cases impossible to distinguish the bodies so characteristically described by Riess, from true micrococci; and I can therefore easily imagine that, according as one wished to find that bacteria were present or absent, the granular constituents of the blood would be regarded as micrococci, or micrococci when present would be regarded as the remains of disintegrated white corpuscles. I have, however, on many occasions examined normal blood and normal tissues by means which prevent the possibility of overlooking bacteria, or of confounding them with granular masses of equal size; and I have never, in a single instance, found organisms. *I have therefore come to the conclusion that bacteria do not occur in the blood, nor in the tissues of the healthy living body either of man or of the lower animals.*

On the other hand, the following objections which have been raised against the assumption that bacteria are the cause of traumatic infective diseases seem to me to be well founded. In order to establish this assertion (that they are the cause of traumatic infective diseases) it would be absolutely necessary *that the presence of bacteria in these diseases be proved without exception, and further that the conditions as regards their*

* Virchow und Hirsch. Jahresber., 1872. Vol. i., p. 252.

number and distribution be such as to afford a complete explanation of the symptoms. For, if in some cases of a certain form of infective disease bacteria be found, while in others of like nature they are absent, and if further the bacteria present be so few in number that it is impossible that a severe disease or a fatal termination could thereby be produced, then of course nothing remains but to regard the irregular appearance of the bacteria as depending on chance, and their small number as insufficient as an only cause of the disease in question; in other words it is necessary to assume the presence of some other agency. The observations hitherto made with regard to the occurrence of bacteria in traumatic infective diseases do not, however, in reality fulfil the necessary conditions.

On account of the difficulties, before alluded to, attending the demonstration of bacteria in the blood, and more especially in the tissues, many of the above-mentioned statements have been received with considerable suspicion,—whether always with justice must remain uncertain,—for the earlier method of investigation is in most cases a groping in the dark, and its results cannot be otherwise than very doubtful.

But, apart from the uncertain results of much laborious work on the bacteria of traumatic infective diseases, the literature of the subject contains a number of statements as to complete absence of bacteria in undoubted instances of these affections. It would serve no good purpose to enumerate here all these negative statements, as their value is even less than that of the positive. One or two may, however, be mentioned as illustrations.

Birch-Hirschfeld* states that negative results with regard to the occurrence of bacteria, especially in cases of spreading (fulminanter) gangrene and putrid infection, are by no means rare.

Orth,† after mentioning that micrococci are present in the blood, particularly in septic diseases, puerperal fever, and diphtheria, expressly states that they are by no means constantly found.

* *L. c.*, p. 1224.

† Compendium der pathol.-anatomischen Diagnostik. Berlin, 1876, p. 111.

Eberth,* who has convinced himself of the frequent occurrence of bacteria in septicæmia, does not regard that disease as caused by an infection of the blood by bacteria alone, because he has seen the most distinct septicæmia without the presence of bacteria in the blood.

Weigert† speaks thus of the occurrence of bacteria:—"However certainly one may refer the development of some diseases to the action of bacteria, yet, on the other hand, there exist a far greater number of morbid processes which would theoretically be considered as mycotic, but in which a conscientious observer cannot discover any traces of bacteric agency."

I have intentionally taken these extracts from the writings of those authors who have shown, by having sometimes obtained positive results, that they understood how to overcome the great difficulties connected with the discovery of bacteria, and whose statements, therefore, with regard to their frequent absence in cases of traumatic infective diseases, deserve especial consideration.

The second circumstance, which seems to me of essential importance in considering this question, *viz.*, that in almost all cases in which bacteria were found the number was strikingly small, has as yet received too little attention.

We do not yet know with certainty how many bacteria are necessary to produce in man definite symptoms of disease, or what proportion per kilogram of an animal is required to cause a fatal result. Undoubtedly some definite relations, varying at most only within narrow limits in consequence of differences in the affected individuals, must exist between the number of pathogenic bacteria and their effect, *i. e.*, the symptoms of the disease. The only disease which may with certainty be affirmed to be of bacteric origin, *viz.*, anthrax, gives us sufficient ground for this supposition. Small animals die after inoculation with anthracic blood more quickly than do large ones, and in animals of the same genus and of like size the fatal termination occurs later when the fluid used contains but few spores or bacilli capable of development than when it is rich in them. The explanation of these facts

* Med. Jahrbb. Vol. 166, p. 185.

† Berl. Klin. Wochenschr., 1877, No. 18.

can only be this, that to kill, *e. g.*, a sheep, more bacilli are requisite than to kill a mouse ; that bacilli or spores being introduced in about equal quantities into both animals at the time of inoculation, the number of bacilli necessary to cause the death of the mouse, being small, is reached more quickly than the larger quantity requisite to kill the sheep ; and further, that in animals of the same species the fatal number of bacilli is longer in being attained when the spores introduced are few in number than when they are injected in large amount.

We learn further from the study of anthrax that the number of bacilli present in the blood must be enormously large before death results. In relapsing fever also, the relations of which to the spirochæta discovered by Obermeier are as yet not sufficiently known, but which nevertheless, on account of the absolutely constant occurrence of these organisms during every attack of fever, must be regarded as in all probability a parasitic disease, the same relation exists between the number of bacteria in the blood and the symptoms of the disease. Of course it must not be assumed that all pathogenic bacteria behave alike in this respect, but it may be concluded from the analogy of anthrax and relapsing fever that a considerable number of bacteria is necessary to produce symptoms. But the observations which have been hitherto made with regard to bacteria in traumatic infective diseases do not in most cases show that this requirement is fulfilled. Mention is generally made of large masses of micrococci on the surface of the wound, which collections, however, can only be regarded as of importance in large wounds; while in internal organs only miliary colonies of bacteria have been found, and these often in small numbers. This result bears no relation whatever to the almost incredible numbers of bacilli in the blood in anthrax. It is therefore only observations which show the presence of a large number of bacteria which can be regarded as affording sufficient explanation of the morbid appearances. Statements no doubt exist with regard to the occurrence of micrococci in the blood and tissues, but unfortunately it is precisely these assertions which for the reasons above-mentioned are the least satisfactory.

A third point remains to be urged against the cogency of the facts known as to the occurrence of bacteria. It is this, that morphologically the bacteria found in the most diverse traumatic infective diseases, and also in other infective processes not in any way connected with wounds, are strikingly similar.

It must at once strike anyone who studies the literature on the subject of bacteria that the two best known bacteric diseases, anthrax and relapsing fever, are notable for the well-marked and easily recognisable form of their parasites; but that in almost all other infective diseases, which apparently stand in close relation with micro-organisms, there exists a remarkable agreement as regards the form, size, arrangement, and colour of the bacteria observed. But, for the very reason that in relapsing fever and in anthrax such marked differences in this respect exist, the similarity of the other pathogenic bacteria must awaken distrust as to the accuracy of the observations and of the assumption that diseases which seem to bear so little relation to each other should, nevertheless, be produced by the same organisms.

Such doubts have been often expressed.

Thus, for example, Birch-Hirschfeld* says, "the morphological characters of the bacteria found in pyæmia, diphtheria, small-pox, and cholera are so similar that the idea naturally arises that identical organisms are being dealt with. But, if this were the case, it would follow that no specific significance could be attributed to these forms. They would have to be regarded merely as parasites of the disease and not as its cause."

To the diseases mentioned by Birch-Hirschfeld a number of others may be added in which micrococci, indistinguishable from each other, were found. Such are erysipelas, puerperal fever, mycosis of the navel in newly-born infants (nabelmycose), hospital gangrene, intestinal mycosis, endocarditis (with or without acute articular rheumatism), primary infective periostitis, scarlet fever, rinderpest, and pleuro-pneumonia. It is, however, impossible that all these diseases can be

* *L. c.*, p. 233.

produced by one and the same parasite, and we must therefore assume either that the micrococci are in reality always the same, in which case they would be merely associated, as an accidental complication, with the diseases enumerated, or that the micrococci—though, on account of their small size, very similar, and indeed apparently the same—are nevertheless different in nature, and consequently capable of giving rise to these diverse results.

In order to show that this latter assumption does not lie beyond the range of possibility, Cohn* has called attention to the apparently similar external and microscopical nature of the sweet and bitter almond, while a great difference exists between them in physiological action. And Virchow,† in the same sense, has referred to the fact that one cannot say with regard to the formative cells of the egg and numerous pathological growths what structure wiil be developed from them, although as compared with bacteria these are truly gigantic in size.

The possibility that the micrococci, in spite of their uniform appearance, may be in reality different, and the true contagium vivum of the disease in which they are found, must assuredly be admitted. But as a practical groundwork, more especially with regard to the prophylaxis and treatment of the traumatic infective diseases, the *possibility* of a contagium vivum does not help us much; we require conclusive evidence that this or that micrococcus, definite in nature and always recognisable under varying conditions by certain characteristics, is the only cause of the disease in question. So long as the existence of a contagium vivum is only a matter of possibility, or even of probability, we cannot avoid taking into consideration in all our investigations, the likewise possible presence of other causes of disease, *e.g.*, of the unknown x of a lifeless disease ferment never yet demonstrated, of the y of the genus epidemicus, and of other unknown quantities. It is, however, apparent that the solution of the problem proposed would thus be rendered in the highest degree difficult, and would be endangered through numerous sources of error—in fact might probably be quite impossible.

* Beiträge zur Biologie der Pflanzen. Vol. I., part 2, p. 135.

† Die Fortschritte der Kriegsheilkunde. Berlin, 1874, p. 33.

If we now look at the facts brought together and the remarks on them, we come to this conclusion, that the frequent discovery of micro-organisms in traumatic infective diseases and the experimental investigations made in connection with them render the parasitic nature of these diseases probable; but that a thoroughly satisfactory proof has not yet been furnished, and can only be so when we have succeeded in finding the parasitic micro-organisms in all cases of the disease in question, when we can further demonstrate their presence in such numbers and distribution that all the symptoms of the disease may thus find their explanation, and finally when we have established the existence, for every individual traumatic infective disease, of a micro-organism with well-marked morphological characters.

Is it then possible to fulfil these conditions in any degree? or have we now, as many microscopists assume, reached the limit of the capabilities of our optical appliances?

This question will, indeed, have often enough occurred to every one who has specially devoted himself to the examination of pathogenic bacteria. It has also occupied my attention greatly, and at once forced itself on me when I commenced these general investigations on bacteria, and saw what great advantages might be obtained by a proper use of microscopic aids in recognising and distinguishing the smallest forms of bacteria with their spores and cilia.

Since that time I have unremittingly attempted to improve the means for the discovery of pathogenic bacteria in animal tissues, because I could not get rid of the idea that the doubtful results of investigations with regard to the parasites of infective diseases might have their foundation in the incompleteness of the methods used.

I shall now, before passing on to the experimental part of my work, describe the method which I have finally adopted as most suitable, and which has led me to positive results.

METHOD OF INVESTIGATION.

Von Recklinghausen's method, in which advantage is taken of the property bacteria have of resisting acids and alkalies, a power not possessed by animal tissues, is still used by most microscopists. If a group of extremely minute particles, characterised by the uniform size of its component granules, does not alter either in acetic acid or in caustic potash or soda, and if there are other grounds for suspecting the presence of bacteria, the problem may then generally be regarded as solved, and the micrococcus found. An error cannot easily occur here, because the appearance of a closely-packed heap of micrococci, the so-called zooglæa, is so characteristic that he who has once had this picture stamped on his mind will recognise it at any time. Incomparably more difficult are the circumstances when bacteria—and this particularly applies to micrococci—are scattered through the tissues either singly or in small loose groups. For now the characteristic appearance of zooglæa no longer aids the observer, and one has solely to depend on the resisting power of the bacteria to alkalies and acids, because there are a great number of minute particles present in tissues altered by disease which may very easily be confounded with organisms. This method, however, of distinguishing by reagents soon shows itself unreliable. Many bacteria, more especially those which are extremely minute, are as readily destroyed or altered by these reagents as are the animal tissues; and, in the latter, indefinite granules often occur which are not removed by acids or alkalies. This method, therefore, cannot do more than demonstrate zooglæa masses.

An attempt has been made to obtain better results by the use of staining fluids, and the one which has been chiefly recommended, simultaneously indeed by several observers, is hæmatoxylin. This is a great step in advance of the first method, especially when it is used in the same way as for staining nuclei. But it is very incomplete, inasmuch as the

hæmatoxylin does not stain rod-shaped bacteria at all, and only colours the spherical so slightly as to prevent their certain recognition when isolated. The use of hæmatoxylin is a great advance on the simple examination of the object with reagents, because when stained the bacteric masses are very much more evident than the rest of the tissue, and one is thus less likely to overlook them or to confound them with other objects. This method, which does not of course exclude examination with reagents, also possesses this great advantage, that the stained preparations can be preserved in Canada balsam, and can thus be used at any time for comparison with others.

Staining with the aniline dyes has yielded still better results than staining with hæmatoxylin. So far as I know, aniline staining, as a means of demonstrating bacteria in animal tissues, was first employed by Weigert.* His method, by the communication of which he has laid me under the greatest obligation, is as follows:—

The objects for examination are first hardened in alcohol. The sections made from these are allowed to lie for a considerable time in a pretty strong watery solution of methyl-violet. They are then treated with dilute acetic acid, the water removed by alcohol, cleared up in oil of cloves, and mounted in Canada balsam.

Instead of methyl-violet other aniline dyes, *e.g.*, fuchsin, aniline-brown, &c., may be used in the same manner.

This is, of course, only a general outline of the method; for the individual tissues, and more especially the different forms of bacteria show so great a variety of result from such treatment that it would be impossible to lay down rules which would be universal, and which would apply to every case. For many objects fuchsin is best adapted; for others the methyl colours are more suitable. Among these latter there exists such a difference in the staining power that the sections must lie in one solution only a few minutes, in another several hours. One must therefore work with a large number of sections at once, and test them as to the most suitable

* Bericht uber die Sitzungen der Schlesischen Gesellschaft für Vaterl. Cultur. Dec. 10, 1878.

staining material and as to the time required for the staining. The experienced investigator will find out after a few attempts what is the most suitable material. The strength of the acetic acid solution is not of much consequence. The best solution is one containing only a small percentage of the acid, and it is well not to allow it to act too long. The other manipulations, such as the removal of water, clearing up and mounting, are exactly the same as in the preparation of other microscopic specimens. One must avoid leaving the sections too long in alcohol or oil of cloves, otherwise the staining material will be washed out by these fluids.

In preparations which are treated in this way only the nuclei of the cells and the bacteria are seen to be stained. The latter as a rule take up the aniline colour, and, in fact, their staining is so marked that the individual bacteria can be much more distinctly recognised than after the use of hæmatoxylin. It is thus very easy to recognize with certainty isolated large bacteria, *e.g.*, bacillus anthracis, in the most diverse tissues, when the preparations have been treated with aniline dyes. As soon, however, as we have to deal with smaller bacteria the method yields uncertain results, and, finally, with the smallest forms becomes quite useless.

In order now to understand how it is that small objects, notwithstanding intense staining, cannot be distinguished at all in the animal tissues, or only with difficulty, one must clearly comprehend the component parts of the microscopic picture. Let us for the sake of simplicity consider only the case of a section of an animal tissue mounted in Canada balsam in the ordinary manner.

If all the constituents of this tissue were colourless, and had the same refractive power as Canada balsam, nothing whatever would be seen. This is, however, not the case. Fibres, nuclei, and many other portions of tissue, differ from Canada balsam in their refractive power, and thus by diffraction of the rays of light passing through them an image consisting of lines and shadows is developed which may be termed shortly the structure picture.*

Let us now suppose a second case, namely, that portions

* Nægeli und Schwendener: Mikroskop. Leipzig, 1877, p. 220.

of the tissue, *e.g.*, cell nuclei and bacteria, were coloured, then the conditions would appear as follows :—With equal refractive power of tissue and Canada balsam, nuclei and bacteria would be alone visible, and that on account of the staining material with which they are impregnated ; we should therefore have a pure colour picture quite different from the structure picture produced by fibres, membranes, &c., but in part coinciding with that, as for example in the case of the nuclei. For the best possible demonstration of bacteria, which are particularly intensely stained by aniline dyes, such a pure colour picture would certainly be the most suitable. The unavoidable structure picture, however, interferes with this.

Large coloured objects, as for example bacillus anthracis, are but little affected in this way as regards distinctness. Only when the section as a whole or when portions of the tissue are very thick (*e.g.*, the intestinal mucous membrane in its whole thickness) may the structure picture become so preponderant, the number of the shadows placed above each other so great, that even the large bacillus anthracis can be no longer easily distinguished. When, however, the bacteria are smaller and thinner, and thus take up less pigment, the bad effects of the structure picture are much more apparent; a broad dark line may then so overshadow some bacteria that their colour picture becomes too weak to make an impression on the eye. In very thin sections, and in those tissues whose structure consists of but few lines and shadows (*e.g.*, subcutaneous cellular tissue, cornea, &c.), very small bacteria may indeed be distinguished with some accuracy. Ultimately, however, a point is reached where the bacteria are so small that the tiny stained granules and threads are hidden and rendered invisible by even the faintest structure shadows. In some particularly favourable places one may indeed suspect the presence of bacteria, but a sure recognition of them and distinction of their form and size is no longer possible.

This difficulty was experienced by me in these investigations. In materials stained in the manner to be subsequently described I easily found large bacteria, and also smaller ones, particularly when they formed accumulations in the glomeruli of the kidney. But now the thought arose, must not bacteria

be present also in the spleen and in the capillaries of the lung? For the spleen was swollen and the blood from the left side of the heart which had just passed through the lungs produced on inoculation on another animal the same fatal disease and the same extremely fine granular accumulations of micrococci in the glomeruli as in the first animal. But in spite of the greatest pains the suspected bacteria could not be found. In the septicæmia of mice, which, as I shall afterwards show, is in the highest degree infective, I was quite unable to demonstrate any micro-organisms. I thus obtained the same incomplete results as the investigators who had formerly studied traumatic infective diseases.

At that time, in attempting to photograph bacteria embedded in Canada balsam, my attention was directed to the fact that the microscopic image consists of a structure and of a colour picture, and I found that the structure picture can be markedly increased or weakened by the nature of the illumination. In this there is nothing new. Every microscopist knows what is the effect of the diaphragm placed below a preparation. A narrow diaphragm not only darkens the field of vision, but makes the structure of the object more apparent; a wide one, on the other hand, renders the whole picture clearer, but makes portions of the structure more indistinct. The difference between narrow and wide diaphragms becomes still more strikingly apparent, when, as in photography, not merely a concave mirror, but a lens or condenser, is used for the illumination, and for this reason, that—particularly with a condenser of short focal distance—the cone of rays illuminating the object is capable of much greater variation. When a narrow diaphragm is placed before the condenser the base of this cone becomes so small that the whole cone may almost be regarded as a bundle of parallel rays of light. The larger, however, the opening of the diaphragm is, so much the larger does the radius of the base of the cone become, its length still remaining the same, but far surpassing as regards the ratio of breadth of base to length that obtained by an ordinary concave mirror. If now we examine a microscopic preparation with an illumination in which the cone of rays is at first narrow, but is gradually made broader, though always remaining of the same

length, we shall at once see that—as indeed cannot be otherwise according to dioptrical laws—the structure picture resulting from diffraction, which picture was most marked when the narrowest diaphragm was used, becomes less and less apparent. In proportion, however, as the structure picture diminishes, does the colour picture become more intense and sharply defined. Thus a method is indicated by which the effects of the structure picture may be in so far obviated that even the smallest stained bodies which are within the optical power of the instrument become distinctly discernible. That is to say, an illumination cone must be used of so wide a base that the appearances resulting from diffraction may be completely removed. I tried numerous different lenses and condensers without finding one which removed the structure picture sufficiently till I fell in with the illumination apparatus suggested by Abbe and made by Carl Zeiss in Jena, which I found to answer my purpose in every way.

This apparatus consists of a combination of lenses, the focal point of which is only some millimetres distant from the lowest lens of the objective system. When this compound lens is placed in the opening of the stage of the microscope, a little deeper than the level of the stage, the focal point coincides with the object to be examined, and the latter thus obtains the most favourable illumination. The angle of aperture is so large that on escaping from the condenser into water the outermost rays are inclined at an angle of almost 60° with the axis, the whole effective pencil possessing thus an angle of aperture of 120°—a greater angle than is given any other condenser.* The rays of light are conveyed to this system of lenses by a mirror which is only moveable round a fixed point in the axis of the microscope. Between mirror and lens, and near the focal point of the former, is a support for diaphragms, which are moveable both laterally and circularly, so that the direction of the illuminating pencil may be altered in any way desired. By the use of diaphragms with larger or smaller apertures the aperture of the pencil may be modified from the largest to the smallest attainable with the apparatus. By lateral displacement of the diaphragm without movement of the mirror,

* Naegeli und Schwendener. *L. c.*, p. 99.

oblique illumination can be obtained, and by shutting off the centre of the opening in the diaphragm the middle of the pencil can be got rid of.

By means of this apparatus the connection before described as existing between structure and colour picture can be made evident in the most simple and convincing manner. Let us suppose that a section of a tissue containing very few bacteria stained with aniline is to be examined by the aid of Abbe's illumination apparatus. At first a diaphragm with a narrow opening is used.* The illumination of the object is then about the same as in illumination with a concave mirror and medium cylinder diaphragm. The field of vision, therefore, appears pretty dark, the structure of the tissues is distinctly marked, more especially do the nuclei of the cells strike the eye as dark bodies with a but slightly pronounced staining of a dark blue or red colour; as regards the smaller granules one cannot ascertain at all with certainty whether they are stained or not, nor can it be made out whether these particles are bacteria or constituents of the tissue. Now let diaphragms with a constantly increasing size of aperture be used one after another. The picture gradually alters in a most striking manner. The dark outlines of the cells and cell nuclei, and the sharp lines of the elastic fibres, walls of vessels and the like become pale and ill-defined; the shadows of the bodies present above and below the visual level disappear more and more; many of the points and granules previously seen, which might possibly have been considered to be bacteria, disappear completely, while, on the other hand, small objects which formerly appeared black are observed to be coloured, and the colour of the nuclei becomes more distinct. The field of vision at the same time becomes clearer. The more the lines and shadows and all the differences between clear and dark disappear, so much the more sharply and strongly do all coloured objects stand out, and so much the more distinctly can one recognise their outlines and minute differences in tone and strength of colour. Finally, when the last diaphragm has

* I have had a set of diaphragms prepared, the openings of which increase constantly one millimetre, thus furnishing all varieties of illumination.

been removed, all the outlines of mere structure have disappeared, the field of vision is uniformly cleared, and only coloured objects can be seen. The clearer the light which one selects for illumination (the best light is that from white clouds illuminated by the sun), so much the brighter and more sharply-defined do these objects appear. It then becomes easy to distinguish among the stained bodies the bacteria of which nothing could previously be seen, or which appeared as dark indefinite granules, rods, &c. And this is the more easy, as there is almost nothing stained but nuclei and bacteria. The outlines and size of the bacteria can thus be recognised, and by their uniform appearance they can be distinguished with certainty from other stained granular masses—*e.g.*, broken down cell nuclei.

A very simple arrangement may serve to demonstrate the action of Abbe's illumination apparatus. This consists of a small glass vessel filled with Canada balsam, in which small coloured and colourless glass beads are placed. Here, therefore, conditions are present similar to those in a stained preparation mounted in Canada balsam. The coloured beads correspond to the stained nuclei or bacteria, the colourless to the parts of the tissue which are unstained. If one looks through the vessel on to a broad sheet of paper placed immediately below it and brightly illuminated with daylight, the colourless beads cannot be seen, while the coloured, on the other hand, are distinct and sharply defined. If now the paper be placed at a greater distance from the glass—that is to say, if the pencil illuminating the pearls becomes longer and its angle of aperture smaller, its base remaining the same—the same appearance occurs as when with Abbe's illumination apparatus diaphragms with smaller openings are used in succession, the colourless beads begin to be gradually visible, acquire more distinct and darker outlines, while the coloured ones become darker; and, finally, the two kinds can hardly be distinguished, the coloured becoming completely masked by the colourless. Microscopists who examine for the first time preparations highly magnified and illuminated with Abbe's apparatus without a diaphragm are generally struck with the unusual appearance, the field being too bright and confused,

although the outlines of the coloured objects are sharply defined. Such observers have been accustomed to the dark field obtained by ordinary illumination with a concave mirror, and they accordingly feel the want of the outline of the tissue structure. For them it is expedient not to dispense entirely with the diaphragm, but to increase the size of the aperture until the stained object under examination appears sufficiently distinct; there will then remain quite enough of the structure picture to enable them to make out the relations of the tissue to the coloured objects.

It is well in general to use, in addition to the examination of the bacteria by means of the pure colour picture, other methods of investigation, such as the observation of the structure of the tissue at the same time, and the examination of the fresh object, with or without the use of alkalies and acids; and I may here expressly mention that I have often made use of these chemical tests as a check, in addition to my chief method of examination.

Although the aniline staining and the use of Abbe's illumination apparatus so markedly facilitate the investigation of pathogenic bacteria, we must not imagine that all difficulties are thus removed and all sources of error shut out. On the contrary, a considerable amount of practice is necessary before one is in a position rightly to utilise these very efficient means. Some of the difficulties which most frequently occur may be shortly alluded to.

As even isolated bacteria do not escape the observing eye, it not unfrequently happens that one meets with organisms which are derived from the fluids used for staining, washing, &c. For even distilled water is almost never free from bacteria. One, however, very soon learns to distinguish these bacteria from others, and to recognise them immediately as accidental impurities.

Further, incipient putrefaction must be suspected whenever isolated bacteria are found in the superficial layers of organs. But the bacteria appearing in putrefaction, at first generally large bacilli,* are so characteristic that they are not easily

* See my paper on "Photographiren der Bakterien." Beiträge zur Biologie der Pflanzen. Vol. 2, part. 3. Photogramm No. 6. Plate xvi.

confounded with the pathogenic bacteria. Nevertheless, it is well to be cautious in drawing conclusions from preparations already containing putrefactive bacteria; indeed it is best not to use such tissues at all. In order to eliminate every risk of confusion with the putrefactive organisms, and to exclude the idea that in arrangement and number the pathogenic bacteria may have undergone alterations after death, I have only used objects for examination which were placed in absolute alcohol immediately after the death of the animal, though in a few cases a delay of some hours has occurred. Consequently I have never found putrefactive bacteria in the preparations obtained in this way. On the other hand, I have seldom failed to find them in preparations obtained from human subjects, although the *post mortem* examination was made ten to twenty hours after death.

I must here draw attention to a remarkable variety of cells which might give rise to confusion with small masses of micrococcus. These are the so-called plasma cells, described and figured by Ehrlich;* flat cells, for the most part situated on the external coats of blood-vessels, and consisting of a round heap of granules grouped around a nucleus. Their behaviour with respect to aniline staining is exactly the opposite of that of all other cells. In the latter only the nucleus is stained; in the plasma cells, on the other hand, only the finely granular plasma is coloured, the nucleus remaining unstained. Now as the granules have exactly the size of many micrococci, the plasma cell presents the appearance of a small micrococcus colony, more especially when the nucleus is indistinct or has disappeared. But the granules are commonly of unequal size. This fact, taken along with the presence of a nucleus and the results of comparison with other similar cells, enables the diagnosis to be easily made. In human tissues these plasma cells are not very abundant, but they occur in great numbers in mice, particularly in the skin of the ear.

If it be wished to exclude entirely all possibility of confusing bacteria with portions of animal tissues, or if it be desired to render the number and distribution of the bacteria in an organ

* Archiv für Microscopische Anatomie. Vol. xiii. 1877, p. 263.

more evident, then the following method may be made use of: —After staining with aniline, the sections are treated with a weak solution of carbonate of potash, instead of with acetic acid. By this means the nuclei and plasma cells—indeed as a rule all animal tissues—lose again the colouring matter, and the bacteria alone remain stained. Large sections, in which the bacteria only are stained by the method just described, form splendid objects for affording a general view.

In microscopical *technique* staining methods play an important part, and many of the most valuable discoveries already made have been obtained by their help. But the full benefit which staining affords in microscopic work can only, as my investigations show, be completely obtained by making use also of a suitable apparatus for illumination.

This has not yet, so far as I am aware, been taken advantage of, and I do not therefore consider it superfluous to recommend my method of illumination for other microscopical investigations in which it is desired to differentiate very small stained elements from others.

With regard to the method of using Abbe's apparatus, I must draw attention to the fact that a sharply-defined picture can only be obtained by the use of such objective systems as have all the zones of the objective aperture properly corrected. The objective systems made by Zeiss are tested by means of Abbe's condenser as to the correctness of the individual zones, especially of the marginal ones. These, and more especially the new oil systems constructed after the designs of Abbe, are therefore thoroughly adapted for the observation of colour pictures. In other systems which I have tried from this point of view, the marginal zones were almost always insufficiently corrected. The only other lenses with which I have obtained well-defined colour pictures were made by Siebert and Kraft.

ARTIFICIAL TRAUMATIC INFECTIVE DISEASES.

I. Septicæmia in Mice.

Mice are especially adapted for experiments on infective diseases, as I previously found in my investigations on anthrax. I therefore attempted to produce artificial traumatic infective diseases in these animals by the use of the method which was followed by Coze, Feltz, Davaine, &c.

Accordingly putrid fluids, *e.g.*, putrefying blood, putrid meat infusion, &c., were injected under the skin of the back in mice. The result of such an injection differs much according to the nature of the putrid fluid, and according to the quantity which is introduced. Blood and meat infusion, which have putrefied for a long time, appear to act less injuriously than fluids which have putrefied for a few days only. Of these latter fluids, as, for instance, of blood which has not putrefied too long, five drops is sufficient to kill a mouse within a short time. In this case marked symptoms may be observed in the animal immediately after the injection. It becomes restless, running about constantly, but showing great weakness and uncertainty in all its movements; it refuses food, the respiration becomes irregular and slow, and death takes place in four to eight hours.

In such a case the greater part of the fluid injected is found in the subcutaneous cellular tissue of the back in much the same condition as before it was injected. It contains bacteria of the most diverse forms, irregularly mixed together, and as numerous as when examined before injection. No inflammation can be observed in the neighbourhood of the place of injection. The internal organs are also unaltered. If blood taken from the right auricle be introduced into another mouse no effect is produced. Bacteria cannot be found in any of the internal organs, nor in the blood of the heart.

An infective disease has therefore not been produced as the

result of the injection. On the other hand, there can be no doubt that the death of the animal was due to the soluble poison, sepsin, which has been shown by the researches of Bergmann, Panum, and various other investigators, to exist in putrid blood. The animal has accordingly died not from an infective disease, but simply from the effects of a chemical poison.

This supposition is confirmed by the fact that when less fluid is introduced into the animal, the symptoms of poisoning which follow are less marked, and are quite absent when one or at most two drops have been injected. After the use of such small quantities of blood, mice often remain permanently without any morbid symptoms. But a third of them, on an average, become ill after the lapse of about twenty-four hours, during which time they have remained apparently healthy. The symptoms which are then present are characteristic and constant, and are in no case preceded by any of the symptoms of poisoning previously described.

Before I describe these symptoms, I must mention that the infection may succeed when even less than one drop of putrid fluid has been used. The less the amount employed in the first instance, the fewer are the animals affected; for example, of twelve animals inoculated in the ordinary manner with one-twentieth to one-tenth of a drop each, only one was successfully infected.

The first symptom in the infected animals is an increased secretion from the conjunctiva. The eye appears dull, and a whitish mucus collects between the lids, and finally completely glues them together. At the same time lassitude sets in, the animal moves little and languidly; as a rule it sits quite still, with its back much bent and its extremities closely drawn up. It then ceases to eat; its respirations become slower, weakness increases more and more, and death comes on almost imperceptibly. Convulsions never precede it (they always do so in anthrax). After death the animal still remains in the sitting posture with its back strongly bent, while, on the other hand, a mouse which has died from anthrax is always found lying on its back or side with its stiffened extremities fully extended. Thus by the position of

the body after death, a fatal result produced by the inoculation of putrefying blood is at once distinguished from that occasioned by inoculation with the material of anthrax. The death of mice infected with putrefying blood occurs forty to sixty hours after the inoculation.

On post-mortem examination there is found at the place of injection or inoculation slight œdema of the subcutaneous cellular tissue. This, however, is often absent, and the internal organs, with the exception of considerable swelling of the spleen, appear quite unaltered.

If one now takes a very small quantity (*e.g.*, one-tenth of a drop) of the fluid of the subcutaneous œdema, or of blood from the heart of such an animal, and inoculates another mouse, exactly the same diseased symptoms occur in the latter animal after the same lapse of time and in the same order as in the former, and death takes place in about fifty hours. From this second animal a third may be infected in like manner, and so on through as many successive animals as one pleases. I have performed these experiments on fifty-four mice and have always obtained the same result. Of these, seventeen inoculations were made in succession; of the others the series of successive infections were less extended.

The certainty with which the infective material can be carried from one mouse to another is here even greater than in anthrax. In the latter, in order to obtain constant results the material for inoculation must be taken from the spleen, because the blood of mice affected with anthrax often contains very few bacilli. In the disease of the mouse produced by putrefying blood it is, on the other hand, a matter of indifference from which organ the material used for inoculation is taken, and even the smallest amount will produce an effect with certainty. It is sufficient, in order to bring about the death of the animal within about fifty hours, to pass the point of a scalpel, which has been in contact with the infected blood, over a small wound of the skin. I have often performed the following experiment:—The subcutaneous tissue of a mouse which had died after inoculation on the tail was touched with a knife on the opposite part of the body to that which had been inoculated, *viz.*, on the head, and with

this instrument a small scratch was made on the ear of another mouse. The animals thus infected died, without exception, of the same disease.

This disease is therefore undoubtedly an infective disease, which, from the result of the post-mortem examination, must be called septicæmia.

The great virulence which the blood of septicæmic mice possesses leads us to suspect that if this disease be a parasitic affection brought about by bacteria, the parasites must be present in the blood, and that in great numbers. But in my first investigations I entirely failed to discover bacteria in the septicæmic blood. Not till I used Abbe's condenser did I succeed in demonstrating their presence with complete certainty, in spite of their minute size.

I examined the blood by a method which I have described in another place, and which in this case yielded good results* (by drying it on a cover glass and then staining with methyl violet).

The blood of the animals which became ill after *injection* of one to ten drops of putrefying blood was found to contain as a rule different varieties of bacteria in small numbers, micrococci, and large and small bacilli. If, however, the animals died after *inoculation* with putrefying or septicæmic blood, small bacilli alone appeared in the blood. This result was invariable, and the bacilli were always in large numbers. These bacilli (see Plate i., fig. 1), which lie singly or in small groups between the red blood corpuscles, have a length of ·8 to 1 mikrm.† Their thickness, which cannot be measured accurately, but only approximately estimated, is about ·1 to ·2 mikrm. In order to establish a comparison with other known bacteria there are represented in fig. 4 specimens of Bacillus Anthracis, magnified to the same degree, from the blood of a mouse, the blood having been dried on a cover-glass and stained in exactly the same manner as the septicæmic blood (the lines of junction of the individual rods come out rather too strongly in the drawing). One often sees the bacilli in septicæmic blood attached to each

* Cohn's Beiträge zur Biologie der Pflanzen. Bd. 2, Heft 3, p. 402.

† 1 mikrm. = ·001 millimetre.

other in pairs, either in straight lines or forming an obtuse angle. Chains of three or four bacilli also occur, but they are rare. They show at first sight a great resemblance to small needle-like crystals, but that they are undoubtedly vegetable bodies is evident, for when septicæmic blood is placed on a concave slide and kept in an incubation apparatus the bacilli grow in the same manner as the bacilli of anthrax, not forming, however, long threads like the latter, but dense masses which consist of isolated bacilli. In some cases I have also seen spores appear in the bacilli. I could not, from want of time, study further the conditions of life and of vegetation of these septicæmic bacilli. I intend, however, at some future period to investigate these. Without the use of staining materials the bacilli can only with extreme difficulty be recognised in fresh blood, even when one is familiar with their form, and I have not been able to obtain any certain evidence as to whether they move or not. Their relation to the white blood corpuscles is peculiar. They penetrate into these and multiply in their interior. One often finds that there is hardly a single white corpuscle in the interior of which bacilli cannot be seen. Many corpuscles contain isolated bacilli only; others have thick masses in their interior, the nucleus being still recognisable; while in others the nucleus can be no longer distinguished; and finally, the corpuscle may become a cluster of bacilli breaking up at the margin—the origin of which one could not have explained had there been no opportunity of seeing all the intermediate steps between the intact white corpuscle and these masses (Plate i., fig. 2).

Starting from the point of inoculation, one can easily see the path by which the bacilli have penetrated into the body. In the subcutaneous cellular tissue in the neighbourhood of the inoculated spot they are very numerous, and at times accumulated in dense masses, as can be best observed in inoculations on the ear. They are more especially numerous on the surface of the cartilage of the ear, and they are here covered with a layer of lymph corpuscles. The latter are also present along with numerous red blood corpuscles in the loose cellular tissue.

The large number of red blood corpuscles which pass out of the vessels leads to the conclusion that an alteration has taken place in the walls of these vessels, and thus it becomes extremely probable that the bacilli grow into the vessels and enter the circulation through spaces in their walls, which permitted the exit of the much larger red blood corpuscles. I have never found these bacilli in the lymphatic vessels. Even in the greatly enlarged lymphatic glands they can only be found in the capillary blood-vessels which run through these glands, not, however, in the lymph spaces. In the loose cellular tissue they often spread widely, and may reach from the ear to the mediastinum; from the back into the cellular tissue of the pelvis. I have not found them free in the cavities of the body. Their distribution in the blood-vessels can be best observed on the diaphragm, the vessels running on the border of the centrum tendineum being selected for investigation. The larger veins (Plate iii., fig. 3, shows a small section of one) contain considerable numbers of bacilli pretty equally distributed, and also numerous small clusters developed in the white blood corpuscles. The bacilli which are free in the interior of the vessels are almost always arranged with their long axis in the direction of the blood-stream, and it is thus evident that they were placed in this position by the flowing blood, and after its stagnation have neither increased in number nor moved. In the capillaries the bacilli congregate, particularly at the points of division, but I have never yet seen a complete obstruction of the smaller vessels produced in this way. The inner wall of the arteries is often thickly beset with bacteria directed lengthwise.

In exactly the same manner are the bacilli distributed in the rest of the vascular system. In the examination of sections of lung, liver, kidney, and spleen, one meets everywhere with vessels containing free bacilli, and with white blood corpuscles with bacilli in their interior. The bacilli are not specially accumulated in the glomeruli; strangely enough, they are not more numerous in the greatly enlarged spleen than in other organs.

The whole morbid process has thus a great resemblance to

anthrax. In both diseases the infective power of the blood is due to the bacilli present in it: as soon as these disappear the disease can be no longer produced by inoculation with the blood. Both diseases are distinguished by the invariable development of exceedingly numerous bacilli. There can thus be no doubt that the bacilli of the septicæmia described here possess the same significance as the bacilli of splenic fever, namely, that they are to be regarded as the contagium of this disease.

As anthrax can be successfully inoculated on different species of animals, I have also tried to infect other animals with the blood of septicæmic mice. Having at my disposal only rabbits and field-mice, in addition to house-mice, I was compelled to limit my experiments to them. In both the attempt had a negative result. At first the rabbits were merely inoculated; afterwards the whole of the blood of a septicæmic mouse was injected subcutaneously into one animal, and finally, in addition to the blood, the lungs, heart, liver, kidneys, and spleen of a septicæmic mouse were introduced under the skin of a rabbit.

These animals did not exhibit the slightest evidence of disease, either locally or constitutionally.

It seems peculiar that even field-mice, which resemble house-mice in size and which can hardly at the first glance be distinguished from them, should possess an immunity from this septicæmia. These animals, however, are also much less sensitive to anthrax than house-mice. I attribute this result to differences in the blood of these closely allied animals, which strike one at once on investigation of fresh blood. In the blood of the house-mouse crystals seldom form, and when they do they shoot out only at the border of the drop of blood in the shape of small rectangular tablets and needles. The blood of the field-mouse, on the other hand, always undergoes changes very soon after removal from the body, all the red blood corpuscles becoming transformed into large regular hexagonal plates either immediately or after adjacent corpuscles have run together, and thus the drop becomes in a short time transformed into a crystalline pulp. But although one could not inoculate the septicæmia of the house-mice on

the two species of animals mentioned, yet it does not at all follow that all other species likewise possess an immunity from this disease. Many animals are in like manner insensible to anthrax, and it would certainly repay one to test as many different animals as possible with regard to their behaviour towards this septicæmia.

II. Progressive Destruction of Tissue (Gangrene) in Mice.

Occurring along with the septicæmic bacillus just described I have sometimes found in mice, after the introduction of putrefying blood, a micrococcus in the neighbourhood of the place of injection. This organism attracted my attention by its rapid increase and by its regular formation of chains. As a rule, when the animal dies of septicæmia after about two days, none of the numerous forms of bacteria which were injected with the putrid blood can be discovered, except the septicæmic bacilli, or it may be a few residual specimens growing with difficulty. It must therefore be supposed that none of the other bacteria injected at the same time find in the body of the living mouse a suitable soil, and that they therefore perish more or less quickly. My attention was thus at once arrested, when in some cases micrococci were found growing in unusual abundance and of constantly characteristic form. They were not present in the blood, and by inoculation with the blood the septicæmic bacilli alone were transmitted. In order to test whether they could be inoculated, it was therefore necessary that the material used should be taken from the neighbourhood of the place of injection. Inoculations carried out in this way were successful in producing both forms of disease and the virulence of the serum from the subcutaneous cellular tissue containing these micrococci was just as marked as that of the septicæmic blood. When the point of a knife which had been well cleaned was merely brought in contact with the subcutaneous tissue at a spot about one centimetre and a half from the place of injection or inoculation, and when with this knife another animal was immediately inoculated, the inoculation was successful on every occasion. Septicæmia was of course always produced at the same time, because the serum used contained also

septicæmic bacilli. The influence of these micrococci on animal tissues and their mode of spreading can be best traced on the ear of a mouse; and it is specially instructive to compare an ear on which only septicæmic bacilli have been inoculated with one into which both the bacilli and the chain-like micrococci have been introduced. In the former ear the cellular tissue is full of red blood corpuscles and lymph cells, so that the bacilli can often be recognized only with great difficulty among the numerous cell nuclei. The other ear presents totally different appearances. Spreading out from the place of inoculation one can see extremely delicate and regular micrococcus chains, here pressed together so as to form thick masses, there arranged diffusely, the individual elements of these chains (Plate iii., fig. 6), as can be estimated from measurements of the longer ones having a diameter of ·5 mikrm. These can be traced almost to the base of the ear, and throughout the part occupied by them all the tissues are markedly altered. As far as the micrococci extend, neither red blood corpuscles, nor nuclei of lymph or of connective tissue cells, can be seen. Even the extremely resistent cartilage cells, and the plasma cells so richly present in the mouse's ear and which are likewise characterised by great resisting power, are pale and scarcely recognisable. All the constituents of the tissue look as if they had been treated with caustic potash; they are dead, they have become gangrenous. Under these circumstances the bacteria develop all the more vigorously. The micrococci penetrate in numbers into the damaged blood and lymphatic vessels, and here and there they fill them so completely that the vessels appear as if injected. Among these the septicæmic bacilli, no longer obscured by nuclei, are seen very distinctly in small groups which at times are very dense and remind one of the "Pilz figures" of the inoculated cornea. While the bacilli can be traced up to the root of the ear, and indeed beyond it, have at the same time increased enormously in the blood, and have ultimately caused the death of the animal, the micrococci, on the other hand, and the destructive process associated with them, have only extended during the same time (within about fifty hours) as far as the vicinity of the

root of the ear. Their limit is sharply defined, as can be seen very well on a longitudinal section of the ear examined with a low magnifying power (twenty-five diameters). (Plate i., fig. 5.) The upper part (*c*), from the tip to *b*, is gangrenous. The larger dark oval or round spots (*d*) are transverse sections of vessels containing masses of micrococci in their interior. The widely-distributed micrococcus chains cannot of course be recognised with this power. It is only in the lower fourth of the gangrenous region that they occur in denser groups, which can be seen as little dark points. Then all of a sudden at *b* appears a densely agglomerated mass of nuclei, forming as it were a wall against the invasion of the micrococci, and this is the limit up to which these organisms may be found. They do not extend, even in the blood-vessels, beyond this line. This wall of nuclei has no great breadth, and immediately beyond it comes the normal tissue. By the use of high magnifying powers it becomes apparent that the micrococci do not reach quite up to the nuclear layer. On the side directed towards the micrococci the nuclei are undergoing destruction. Numerous fragments of irregular shape, constantly becoming smaller, form the upper limit of the wall of nuclei, and when this region is reached, in examining the preparation, we may be sure that we are in the neighbourhood of these organisms. There almost always remains between the last remnants of the nuclei and the micrococci a line of considerable breadth consisting only of gangrenous tissue, in which neither micrococci nor nuclei can be found. It is seldom that the micrococci extend into the disintegrating nuclear layer.

These appearances lead us to the conclusion that the action of these micrococci in causing the gangrene is somewhat as follows :—Introduced by inoculation into living animal tissues, they multiply, and as a part of their vegetative process they excrete soluble substances which get into the surrounding tissues by diffusion. When greatly concentrated, as in the neighbourhood of the micrococci, this product of the organisms has such a deleterious action on the cells that these perish and finally completely disappear. At a greater distance from the micrococci the poison becomes more diluted and acts less intensely, only producing inflammation

and accumulation of lymph corpuscles. Thus it happens that the micrococci are always found in the gangrenous tissue, and that in extending they are preceded by a wall of nuclei which constantly melts down on the side directed towards them, while on the opposite side it is as constantly renewed by lymph corpuscles deposited afresh.

These observations refer to inoculations with fluid containing both micrococci and bacilli, and it might have been supposed that the septicæmic bacilli were necessary forerunners of the micrococci, that they must to a certain extent prepare the way for them. I therefore attempted, by various means, to separate these parasites from each other. Thus, at one time a considerable quantity, at another only a little of the fluid was used for inoculation, or again it was taken at different distances from the point of inoculation, or, lastly, the parts of the body to which it was applied were varied as much as possible. But all this was of no avail. Either pure septicæmia or septicæmia along with progressive gangrene was obtained, never the latter alone. Chance led me to the proper method. A field-mouse—which, as I formerly pointed out, possesses an immunity from septicæmia—was inoculated with septicæmic bacilli and chain-like micrococci. The experiment was made in the expectation that neither parasite would develop. This expectation, however, was not fulfilled, for, though the bacilli as usual underwent no development, the micrococci increased and spread in exactly the same manner as has been described in the case of the house-mouse. Beginning at the place of inoculation on the root of the tail, the gangrene spread onwards along the back, passing deeply among the dorsal muscles, and downwards on both sides to the abdominal wall. The animal died three days after the inoculation. The parts affected with the gangrene were partially denuded of epidermis and hairs, and contained chain-like micrococci in extraordinary numbers. The same micrococci were also found on the surface of the abdominal organs, although there was no visible peritonitis. The blood and the interior of the organs were, on the other hand, quite free from them. From this animal other field-mice, and from these again house-mice in various successive series were subsequently injected, and

always with the like result, *viz.*, that only chain-like micrococci and, in their train, progressive gangrene were obtained.

III. Spreading Abscess in Rabbits.

Coze and Feltz, Davaine, and many others have obtained in rabbits, by the injection of putrid blood, an infective septicæmic disease. I have therefore repeated their experiments. I have not, however, succeeded in producing the effects described by Davaine, but I observed—what others who have made similar experiments on rabbits have already noticed—that in these animals the formation of an abscess constantly increasing in extent may occur in the subcutaneous cellular tissue without any general infection taking place. Such animals have at first no symptoms of disease; a flat lentiform hard infiltration at the seat of the injection is all that can be observed. After several days this hardness extends in all directions, chiefly downwards, especially towards the abdomen and anterior extremities. The animal at the same time emaciates and grows feeble, and dies in about twelve to fifteen days after the injection.

The post-mortem examination shows the presence, in the subcutaneous tissue, of extensive flat abscesses with cheesy contents; their walls bulge in various directions, though the whole remains a single cavity. There is also an extreme degree of emaciation, but no alteration in the peritoneum, intestine, kidneys, spleen, liver, heart, or lungs. In the blood the white corpuscles are greatly increased in number, but no bacteria can be found. The cheesy contents consist of a finely granular material, and scattered about in this are nuclei undergoing disintegration, but no bacteria can be definitely made out. Here, then, we have appearances similar to those often found in man, and much used as an argument against the parasitic nature of such morbid processes. I refer to abscesses resulting from phlegmonous inflammation which must be regarded as infective in their origin, but in which no micro-organisms have been found.

When, however, portions of these abscesses are hardened and examined in sections, the surprising result is obtained that, though bacteria are not present in their contents, their

walls are everywhere formed by a thin layer of micrococci united together into thick zooglœa masses. These organisms are the smallest pathogenic micrococci which I have as yet observed. In some places I was fortunate enough to find them arranged in rows, and thus was able to measure them; and I ascertained that they were about ·15 mikrm. in diameter (this is of course only an approximate measurement). From the form and character of the zooglœa masses surrounding the abscesses it follows that these masses stand in the most intimate relation to the contents of the abscesses; that, in fact, the contents are constituted by the zooglœa masses and the dead portions of tissue enclosed by them. This process takes place as follows:—The micrococci grow only in masses which, at the periphery of the more or less lentiform abscess, differ in arrangement from that which they assume on its upper, and more especially on its under surface. The margins of the abscess extend into the loose meshes of the subcutaneous cellular tissue, where the micrococci find the least resistance to their extension, and accordingly surround the abscess in thick cloud-like masses (Plate i., fig. 5). The cellular tissue in the immediate vicinity is more or less richly filled with nuclei (*c*), between which one can see small isolated micrococcus colonies (*b*, *c*)—forerunners, in fact, of the main zooglœa masses. The smallest colonies which can be found seem, from their general form and their radiating pointed processes, to be present in the canaliculi of the cellular tissue. I have not been able to demonstrate any connection between these micrococci and the connective tissue corpuscles, such as is observed in the inoculated cornea. On the wall of that part of the abscess which is directed towards the deeper structures, and where the dense fascia opposes the extension of the organisms, they cannot develop so luxuriantly as at the borders of the abscess. On the contrary, the groups are here small and flattened (Plate iii., fig. 7), and only occasionally send out processes into the layers of the cellular tissue beneath, this tissue being in these situations interspersed with nuclei. An appearance which is quite characteristic can be observed when the zooglœa masses are examined more closely. Their outer borders, by which I understand the parts of the zooglœa masses which are

directed towards the healthy cellular tissue (fig. 8, *a*), are stained by the aniline fluid of an intense dark colour, and the individual micrococci can be distinctly made out. In the small, and apparently young colonies more especially (fig. 8, *b*, *c*, *d*, and fig. 7, *b*) the micrococci are uniformly coloured. But on passing towards the interior of the abscess the staining of the zooglæa becomes less marked, the individual micrococci can be no longer accurately defined, they become more and more finely granular, and ultimately form an almost homogeneous mass which no longer takes the colouring matter (fig. 8, *g*).* Still nearer the abscess cavity are found pale unrecognisable masses derived from the zooglæa (fig. 7, *d*) intermixed with the detritus of the nuclei (fig. 7, *e*, and fig. 8, *f*); and the cheesy contents of the abscess are composed of these two materials alone — the dead zooglæa and the remnants of the nuclei, the former being present in largest amount. I have called these unstained masses *dead* zooglæa for the following reasons:—In the first place, this explanation suggests itself so naturally, and seems such a necessary deduction from direct observation and from the comparison of the small micrococcus colonies found in process of growth with the large zooglæa masses which have completed their vegetative life, that no special proof is required for it. One might fairly compare this growth of the micrococci on the one side and their death on the other with the vegetation of Sphagnum. Other considerations further indicate that, when bacteria are no longer stained by aniline, it is a certain sign of their death. The form of bacteric vegetation described here deserves the greatest attention, for it is evident how easily in similar cases the narrow line of bacteria might be overlooked, even though the latter were still in full growth and easily recognisable. Similar circumstances also apparently occur in human infective diseases. Thus Klebs† found in endocarditis that the micrococci deposited on the aortic valves were dark-coloured on the surface, while in the deeper parts

* The micrococci in figs. 7 and 8 have been drawn too large in parts, more especially towards the interior of the zooglæa.

† Archiv fur experimentelle Pathol. und Pharmak. Bd. IX., p. 72 (Taf. II., fig. 3).

they became paler, and finally quite disappeared, passing into a homogeneous mass.

In order to ascertain whether the morbid process here designated as progressive abscess formation could be transmitted from one animal to another, rabbits were injected with blood taken from others which had already died of this disease. These injections produced no effect. A small quantity of the cheesy contents of the abscess was now taken, diluted with distilled water, and injected under the skin of a rabbit. There resulted exactly the same abscess-formation in this animal as in the first. The abscesses spread in the same manner as described in the former case, and caused the death of the animal experimented on in a week and a half. From this animal the disease was conveyed to a third, and so on through several in succession.

It was thus demonstrated that the disease is not merely occasioned by the injection of a considerable quantity of putrefying blood, but is of a decidedly infective character. The assumption made above that the micrococci in the cheesy contents of these abscesses are dead, does not appear in keeping with this result of inoculation. This apparent contradiction may, however, I think, be cleared up, for it is very probable that these micrococci, like other bacteria, form resting spores (Dauer-sporen) after the expiration of their vegetative life, and that these bodies, just like the spores of bacillus, are not stained by aniline, and therefore remain invisible in Canada balsam. The infection in the case referred to would be brought about by such spores.

IV. Pyæmia in Rabbits.

Having failed in various attempts to produce a general infection in rabbits by the injection of putrid blood, I tried the effect of other putrid fluids.

A piece of a mouse's skin about a square centimetre in size was macerated for two days in thirty grammes of distilled water, and a syringeful of this fluid was injected subcutaneously into the back of a rabbit. This animal remained for two days free from any noticeable symptoms of disease, then it began to eat less, became gradually weaker, and died one hundred and five

hours after the injection. A post-mortem examination was at once made, and there was found a flat, purulent (not cheesy) infiltration in the subcutaneous cellular tissue, extending from the point of injection as far as the hip behind and the linea alba below. In the abdominal wall the yellowish infiltration extended in parts through the abdominal muscles, and even to the peritoneum. The latter was dull and in many places covered with delicate whitish clots. In the peritoneal cavity a small quantity of turbid fluid was found. The intestines were glued together by white fibrinous masses. The liver, stomach, and spleen were covered with thin white layers of fibrin, and the spleen was much enlarged. The liver, after the removal of the deposit on it, presented a greyish mottled appearance, and showed on section grey wedge-shaped patches; its borders were also in parts of a grey colour. In the lungs were found some dark red patches about as large as a pea, devoid of air. As regards the remaining organs no alterations could be detected, not even in the heart.

A syringeful of blood taken from the heart of this animal was now injected under the skin of the back of a second rabbit. The latter died in forty hours. The result of the post-mortem examination was essentially the same. The infiltration in the neighbourhood of the place of injection was, however, more œdematous, and the cellular tissue was besprinkled with small extravasations of blood; the peritonitis was less advanced; on the small and large intestines a few small subserous extravasations of blood were present; and in the lungs and the liver were metastatic deposits similar to those found in the first rabbit.

I had therefore without doubt a general infective disease to deal with. Indeed it was possible that it might be the same affection as had been obtained by Coze and Feltz, and by Davaine, from injections of putrid fluids into rabbits, and which had led them to their observations on the increasing virulence of septicæmic blood when transmitted through a series of animals in succession. I therefore resolved to carry out a series of experiments similar to those already performed by Davaine.

To furnish a general view of the results of these experiments I shall arrange them in a tabular form :—

Rabbits.	Fluid injected.	Quantity of the same.	Death.
I.	Maceration fluid.	10 drops.	In 105 hours.
II.	Blood of I.	10 ,,	In 40 ,,
III.	Blood of II.	3 ,,	In 54 ,,
IV.	Blood of III.	1 ,,	In 92 ,,
V.	Blood of IV.	$\frac{1}{10}$,,	In 125 ,,
VI.	Blood of V.	$\frac{1}{1000}$,,	Remained well.

The blood was diluted in the same manner as had been done by Davaine in his experiments. In order to obtain the one-thousandth of a drop for injection, a drop of blood was mixed with one hundred drops of distilled water; of this mixture one drop was again added to one hundred drops of distilled water, and of the solution so obtained (the blood being now diluted ten thousand times) ten drops were injected. The table comprises only a few experiments, but the relation between the quantity of blood injected and the time which elapsed before death is so constant that it cannot be accidental. There was no indication of increasing virulence of this blood when inoculated into several animals in succession. The less the quantity of blood injected, the longer the time which elapsed before death occurred, and where the quantity was reduced to the one-thousandth of a drop no result followed. I do not say, however, that the infective power of the blood was entirely abolished by reducing the quantity to one-thousandth of a drop, for this experiment was performed on one animal only, and it might very possibly have happened that if several animals had been injected at the same time with the same amount, one or other would have become ill, or even have died. But it follows from the table that when the quantity is small the effect is delayed, and that when very minute the result is uncertain, or even negative. These results can only be explained on the supposition that the blood always contains a like quantity of undissolved infective particles, and that these particles must have increased to a certain number before they can cause death.

For, an infective material in solution, which is active in quantities varying from ten drops to one-tenth of a drop, must

also be infective under all circumstances, even when reduced to a thousandth of a drop; but death would take place at a correspondingly later period.

If, however, the infective material be supposed to be insoluble, and if a certain quantity is always necessary to destroy a rabbit, then the explanation of the fact that the result is more and more delayed in proportion as the injected blood is diluted is at once evident. For the more the blood is diluted the fewer bacteria does each drop contain, and if fewer bacteria be introduced into the animal experimented on, longer time must of course elapse before these have attained the number necessary to cause the death of a rabbit than when the quantity at first injected was large. If the blood be yet further diluted, a time will finally come when in the quantity of blood used for injection, say ten drops, there will not with certainty be a single bacterium, or at least a number sufficient for infection. Then the result becomes a matter of uncertainty.

Let us now see how the facts furnished by microscopic investigation coincide with this explanation.

But first I must mention that the post-mortem appearances found in the last three rabbits were, with some unimportant variations, the same as in the first two, *viz.*, local purulent œdematous infiltration of the subcutaneous cellular tissue, metastatic deposits in the lungs and liver, swelling of the spleen, and peritonitis. These appearances harmonize so completely with those commonly designated as pyæmia, that I do not hesitate to use that term for the disease under consideration.

On microscopic examination micrococci are found in great numbers everywhere throughout the body, and more especially in the parts which have undergone alterations visible to the naked eye. These micrococci are for the most part single or in pairs, and their measurement is therefore difficult. Ten measurements of pairs of micrococci differed but little from each other, and gave ·25 mikrm. as the average diameter of a single individual. As regards size, therefore, they stand midway between the chain-like micrococcus of the progressive gangrene of the tissues and the zooglæa-forming micrococcus of the cheesy abscesses of rabbits. Their relation to the

blood-vessels can be best seen in the renal capillaries, and I have therefore selected a small vessel from the cortex of the kidney for delineation (Plate v., fig. 9). It is impossible in a drawing to represent their relative size correctly, and it was necessary to draw them here on a somewhat larger scale than the micrococci of figs. 7 and 8. In the interior of the vessel at *c* is a dense deposit of micrococci adherent to the wall, and enclosing in its substance a number of red blood corpuscles. This mass would probably have very soon filled the calibre of the vessel, for fresh blood corpuscles are constantly being deposited upon it, and these become surrounded by delicate offshoots from the mass of micrococci. From this we may conclude, either that the micrococci have of themselves, owing to the nature of their surface, the power of causing the red blood corpuscles, to which they adhere, to stick together, or that these organisms can occasion coagulation of the blood in their vicinity, and thus the formation of thrombi.

The manner in which these micrococci, as it were, spin round the blood corpuscles and enclose them, seems to me to be quite characteristic of this particular form. Such partial or complete thrombus formations occur in the renal vessels in many places, particularly in the glomeruli, where individual capillary loops may be found completely blocked by micrococci. But even in these thick zooglæa-like masses one can still recognise the clear circles due to the enclosed red blood corpuscles. As a rule, however, only small groups of micrococci are met with (as in fig. 9, *b*). They were found arranged in this manner surrounding and glueing together a small number of blood corpuscles, in the capillary vascular system of all the organs examined, as, for example, in the spleen and in the lungs. In the larger vessels also groups of considerable size are formed, and I am disposed to believe that the large metastatic deposits in the liver and in the lungs do not arise by gradual growth of a mass of micrococci, as in fig. 9, but by the arrest of large groups of micrococci and of the clots associated with them formed in the manner described in the circulating blood; in other words, by true embolism. In the metastatic deposits an extensive development of

micrococci occurs, and these are not confined to the vessels, but invade the neighbouring tissues. Micrococci in pairs are pretty equally distributed over the surface of the abdominal organs. Masses of micrococci do not form in the peritonea cavity; and the small flakes of pus in the peritoneal fluid and the fibrinous deposits infiltrated with pus cells present on the surface of the abdominal organs, contain micrococci only uniformly distributed, or at most collected into small groups.

In the neighbourhood of the place of injection the subcutaneous cellular tissue is infiltrated with extensive flat collections of pus, which are surrounded by micrococci more or less numerous, but never in the form of zooglæa. They also surround the subcutaneous veins of this part, which are much distended with blood corpuscles, and their presence in the walls of the vessels and their passage through these walls into the interior can be seen in many places. No micrococci were found in the lymphatic vessels or in the neighbouring lymphatic glands, which were, however, greatly swollen.

Comparing the results of the microscopic investigation with the before-mentioned effects of the injection, we find that they are in complete unison.

In the experiments the blood was taken from the heart, and with reference to the cause of its infecting property we have only to consider the state of the blood in the larger vessels. This, as already mentioned, contains numerous micrococci. The first part of the assumption that the infective particles were bacteria is thus proved. If, however, these underwent the same growth in the blood, as the septicæmic and anthracic bacilli, they must become as numerous in the blood as the latter, and the virulence of the blood would thus be much greater than it was in reality found to be. But, as we have seen, the micrococci of pyæmia behave differently in this respect from the organisms of septicæmia and anthrax. As soon as they come in contact with the red blood corpuscles the latter stick together and form larger or smaller clots in the blood. They can thus no longer pass through the minute capillary networks, like the bacilli which move freely among the red corpuscles, but are

arrested in the larger or smaller vessels. From the point of infection fresh micrococci will no doubt constantly pass into the blood, and also individual micrococci will become detached from these small thrombi and emboli and mix with the blood stream. Nevertheless, their total number in the circulating blood cannot exceed a certain point, because they are very soon deposited somewhere. Thus we have a simple explanation of the fact that the number of micrococci present in the body of the animal experimented on constantly increases, and, finally, apart from the disturbances of circulation produced by them, become sufficiently numerous to cause the death of the animal. Nevertheless, the quantity present in the cardiac blood continues pretty uniform, and is so small as to have an uncertain action when the thousandth part of a drop is used.

5. Septicæmia in Rabbits.

After injection of putrid infusion of meat into rabbits, I have twice obtained a general infection of another sort in which metastatic deposits do not occur, and which I would therefore describe, in contrast to the foregoing, as septicæmia. This infusion, like the putrid fluids used in the earlier experiments, contained numbers of bacteria of the most various forms. When injected under the skin of the back of a rabbit it produces an extensive putrid suppuration of the subcutaneous cellular tissue, and the animal dies in three days and a half. At the ichorous spot, which must, on account of its size, be looked upon as the immediate cause of death (owing to absorption of poisonous materials in solution), the same variety of bacteric forms was present as in the meat infusion. At the border of this spot the cellular tissue was infiltrated with a slightly turbid watery fluid, which contrasted strikingly with the brownish stinking ichor in the vicinity of the place of injection. In this œdema fluid great numbers of micrococci of considerable size and of an oval form were almost the only organism observed. In the blood also similar micrococci were found, though only in small numbers. Further, in the papilli of the kidney and in the greatly-enlarged spleen some

of the small veins were completely blocked for short distances with these oval micrococci.

Two drops of this œdematous fluid were now injected under the skin of the back of a second rabbit. The animal died in twenty-two hours, and here, in the neighbourhood of the place of injection, not a trace of pus could be observed. On the other hand slight œdema, with a streaky whitish appearance of the subcutaneous cellular tissue, extended from the point of injection to the abdomen. In this œdematous cellular tissue lay numerous flat extravasations of blood half a centimetre in breadth, the vessels around these being very greatly distended. The muscles of the thigh and of the abdominal wall were also interspersed with small extravasations. In the heart and lungs no alterations were found. In the peritoneal cavity no fluid was present, the peritoneum being unaltered and the coils of intestine not glued together. But the surface of the intestine, in consequence of a number of small subserous extravasations presented an appearance as if injected here and there with blood. The spleen was also very considerably enlarged.

In this second animal the oval micrococci were alone present in the œdematous cellular tissue, all the other bacteria having disappeared. The number of these organisms was very considerable, many of the small veins being completely filled with them. In the hæmorrhagic spots were small veins, which were here and there distended with micrococci, thus presenting spindle-shaped dilatations, which had at parts burst, the micrococci having thus escaped in large numbers into the surrounding cellular tissue. This appearance could be particularly well seen in the muscles of the thigh.

In the pulmonary capillaries the micrococci were not very numerous; they were scattered through the blood singly or in pairs, and occasionally in small groups. In the kidneys these organisms were present in much larger numbers. The great majority of the glomeruli seemed enlarged, as if swollen; their capillary loops were increased in size and distended with red blood corpuscles. The other glomeruli were smaller than usual, the nuclei of their capillary walls being closer together, so that they presented an appearance as if they had been compressed.

In all the enlarged glomeruli, without exception, more or less extensive deposits of oval micrococci were present. These were arranged in longitudinal series and also side by side, so as to form a single layer, covering the inner wall of the capillaries for short distances, but never embracing the whole circumference. The micrococcus colonies thus presented the appearance of short, slightly-curled, trough-like pellicles. In other places the vascular loops were completely distended, and there were also present all transitions from these dense obstructing masses to the small loose colonies and the single micrococci (Plate v., fig. 10).

In the compressed glomeruli colonies were very exceptionally present, and then only of small size. Isolated obstructing micrococcus masses were also present in the vascular capillary network of the medullary substance. They were also present in an isolated form in almost all the vessels. Accumulations of whitish corpuscles in the neighbourhood of the micrococci and alterations in the epithelial cells of the uriniferous tubules were not observed. The micrococci were never seen in the interior of the uriniferous tubules. The spleen contained loosely-arranged micrococcus colonies scattered about in the capillaries in moderate numbers, and also isolated dense deposits which distended the small vessels at the border and in the interior of the Malpighian corpuscles for short distances.

In the capillaries surrounding the intestinal glands numerous obstructing micrococcus masses were present (Plate i., fig. 11). At many points these were so extensive that branching accumulations were seen consisting entirely of these organisms.

The liver, like the lungs, contained no great accumulation of micrococci.

The largest diameter of an isolated micrococcus was ·8 to 1·0 mikrm.

These organisms differ from the micrococci of pyæmia very markedly as regards size, and in most other points. Thus they never enclose the blood corpuscles, even when they have accumulated in large numbers in the interior of the blood-vessels. They rather push them on one side. They do not cause coagulation of the blood, and thus emboli do not occur.

Only in one point do they resemble the pyæmic micrococci, namely, they do not show an increasing virulence when inoculated into a series of animals in succession.

Thus a syringeful (ten drops) of blood taken from the heart of the second rabbit was injected subcutaneously into a third rabbit. This animal died in thirty-six hours, the naked eye and microscopic appearances being exactly the same as in rabbit No. 2.

From rabbit No. 3 two drops of blood were injected into a mouse, and one drop into a rabbit.

The mouse died in thirty-seven hours; the rabbit remained unaffected.

The blood and all the organs of this mouse contained these oval micrococci, just as in the rabbit. A second mouse was then inoculated with blood taken from this mouse's heart, the operation being performed in the following manner:—The point of a scalpel was dipped in the blood of the heart, and about one-tenth of a drop was put into a small pocket-like wound on the root of the tail. This animal remained healthy.

On a second occasion I have, by injection of putrid meat infusion, obtained in a rabbit the same septicæmic process with the same oval micrococci.

Here, also, the disease could only be transmitted to other rabbits when at least five to ten drops of blood were used for inoculation.

6. Erysipelas in Rabbits.

Not only were large quantities of putrid fluids *injected* into rabbits, but various attempts were also made to produce disease by *inoculation* with different putrefying materials. These were not successful. In one case, however, after inoculation of the ear of a rabbit with mouse's dung softened in distilled water, redness and swelling occurred and spread slowly downwards from the point of inoculation. This redness extended on the fifth day as far as the root of the ear. When held up to the sunlight the ear which had not been inoculated appeared unaltered, only the chief blood-vessels

being seen, while the inoculated ear, similarly illuminated, presented a uniformly dark red appearance, the individual vessels being no longer recognisable. It was thicker, and at the same time had become more fluid than the other, its point being bent and hanging down in consequence of its weight. The animal was, moreover, evidently ill, and died on the seventh day.

No effect was produced on another rabbit by injecting into it blood taken from the first. Unfortunately an attempt was not made to transmit the morbid process by inoculation with material from the ear of the affected rabbit to that of another.

Neither in the blood nor in the internal organs of this rabbit were any alterations found worthy of mention, notably no bacteria. The state of matters in the ear was, however, so remarkable, and bore so unmistakably the characters of a parasitic disease, that I have considered it right, although the infective nature of the disease was not directly demonstrated, to give here a description of the affection.

In transverse sections of the ear the blood-vessels were seen to be markedly dilated, full of red corpuscles, and surrounded by the nuclei of numerous white corpuscles. These nuclei were more numerous towards the cartilage of the ear, and on its surface they formed a pretty uniform dense layer. Between this layer and the cartilage cells were seen small fine rods arranged at pretty equal distances, which rods were distributed parallel to the cartilage in the dense cellular tissue which lies immediately outside the cartilage cells. In many places only single rods were seen; in others several were present, being arranged parallel to each other; while again, thickly interwoven clumps of these same rods were found, and that in parts where the white corpuscles were somewhat more thickly accumulated than elsewhere. These rods were present nowhere except close to the cartilage. Longitudinal sections were accordingly prepared which showed very distinctly the distribution of the rods on the surface of the cartilage. Figure 12, Plate i., is drawn from such a section. The large round bodies (*c*) are the nuclei of large flat cells, under which lie the cartilage cells. On this layer of flat cells there is a thick network consisting of bacilli,

and outside these bacilli, indeed in fact covering them, are the nuclei (*b*), of the white corpuscles of which, however, only a small portion remains in the section. In many places the bacilli form more or less round dense clumps (as shown in fig. 12 *a*) which look like a pad of hair. From these clumps long rows of bacilli, in which the organisms become fewer and fewer, radiate in all directions. This reminds one of the peculiar, often starlike, figures which the bacillus anthracis forms when inoculated on the cornea of a living rabbit.* This network of bacilli extended over the whole cartilage of the ear on both surfaces. As the morbid process could be traced in its extension from its origin at the point of inoculation over the whole ear, and as throughout the whole limit of the process these bacilli were present, and as, further, the signs of inflammatory reaction were most marked in the immediate neighbourhood of these organisms, I consider it indubitable that they were the cause of the disease. I have never observed any formation of spores in them. They vary much in length. One rod, in which I could with certainty only distinguish two joints, was 3 mikrm. in length. The longest rod, consisting of six or seven joints, was 9—10 mikrm. in length. They are about ·3 mikrm. thick (the bacillus anthracis may be as long as 20 mikrm. and as thick as 1 to 1·25 mikrm.,—that is to say, about twice as long and three or four times as thick as the bacilli of the rabbit's ear).

* Frisch, *l. c.*, Pl. i., fig. 3, and Pl. ii., figs. 9 and 10.

ANTHRAX.

The numerous investigations which have been made with regard to anthrax have almost all had reference to the behaviour of the bacillus anthracis outside the animal body. With regard to their numbers in the blood the conclusions have, as a rule, been drawn from blood taken by chance from any part of the body. No observations have yet been published as to the number of bacilli really present in the body, and as to their distribution in the vascular system.

In order to supply this deficiency, and because the bacillus anthracis behaves so like the septicæmic bacilli (thus being useful for comparison with the other pathogenic bacteria here described), I have examined rabbits and mice which have died of inoculated anthrax in the same manner as I did those killed by artificial traumatic infective diseases.

The staining of the bacillus alone, as obtained by treatment of sections stained in methyl violet with carbonate of potash, here proved of the greatest service. The mucous membrane of the stomach and intestine can, for example, be so prepared for examination by this method, that even with low powers the bacilli may be seen in all the vessels. In like manner sections of lung, liver, and kidneys, furnish extremely distinct and instructive preparations.

Although I had often previously examined the blood of animals suffering from anthrax, and had thus formed a high estimate as to the number of bacilli present in the body of an anthracic animal, yet I was quite surprised when I saw for the first time sections and portions of organs stained in this way, as *e.g.*, the intestinal mucous membrane and the iris of a rabbit. When magnified fifty diameters such a preparation presents at the first glance an appearance as if a blue colouring-matter had been injected into the vessels. Each intestinal villus is permeated by an exceedingly delicate blue network; in the mucous membrane of the stomach all the capillary network surrounding the gastric glands is stained

blue; in the ciliary processes each projection is injected, and a spiral vessel stained of a dark blue colour leads from thence to the iris and breaks up into a fine blue network with loops directed towards the edge of the iris. The liver and lungs, and the glandular structures, such as the pancreas and salivary glands, are completeiy permeated by the same blue vascular network. Indeed there is no organ which is not more or less injected with the blue mass. It is, however, very striking that this injection is only present in the capillary vessels. All the larger vessels, even the arteries and veins of an intestinal villus, are either not at all stained or have but a light blue streak in their interior, and that only here and there. When magnified 250 times one can see that the blue capillary network is composed of numerous delicate rods (Plate iii., fig. 13), and when a power of 700 diameters is used it is found that the apparent injection is nothing more or less than the bacillus anthracis, stained dark blue, and present in incredible numbers in the whole capillary system. In the other vessels, especially in the larger ones, often only a single bacillus may be met with at long intervals, or they may be quite absent. Here, therefore, we have a striking proof of how little value are conclusions drawn in traumatic infective diseases from the examination of a drop of blood taken from a blood-vessel by chance; for one might quite well take a drop of blood from the heart and find no micro-organisms in it, or one might readily overlook the few which might be present, and that although the capillary system abounds in them.

The distribution of the bacillus anthracis in the capillaries is not, however, quite uniform. There are fewer in the brain, in the skin, in the capillaries of the muscle, and in the tongue than elsewhere; on the other hand, in the liver, lungs, kidneys, spleen, intestines, and stomach, they are always present in the enormous numbers above described. The spleen, to which the disease owes its name, is not distinguished above the other organs which have been mentioned by a greater number of bacilli. In the capillaries themselves the bacilli accumulate in largest numbers at the point farthest removed from the nearest afferent artery and the efferent vein;

that is to say, where the arterial capillaries join the venous, the aggregate diameter of the vessels being there the broadest and the blood stream flowing the slowest. In the intestinal villus this spot is at its apex and the neighbouring part of the periphery; in the liver it lies midway between the ultimate twigs of the hepatic vein and of the vena portæ. Among the points where the bacilli accumulate in greatest numbers are also the glomeruli of the kidney, which become for the most part transformed into clumps of bacilli. It by no means unfrequently happens that from the presence of the rapidly increasing bacilli at the places mentioned, chiefly in the glomeruli, intestinal villi, mucous membrane of the stomach, salivary glands, and pancreas, individual capillaries become torn, and blood with the contained bacilli is extravasated. This occurs most frequently in the glomeruli. Many of these burst and the bacilli pass into the uriniferous tubules. They do not, however, extend far into these; at least I have only found them in the commencement of the convoluted tubules, in which they form long threads interwoven with each other. I have never seen bacilli in the straight uriniferous tubules.

The facts which I have described are those met with in rabbits. Mice, which I have often investigated, behave essentially in the same manner. In the latter animals, however, the spleen is more especially the seat of the bacilli; then come the lungs, and last of all the kidneys. The contrast between the very large numbers of bacilli in the capillaries and their small quantity in the large vessels is even more striking in the mouse than in the rabbit.

I have also had an opportunity of examining the lungs, liver, spleen, and kidneys of sheep which had died of anthrax, and I found here also that the bacilli had the same relations as regards numbers and distribution as in the rabbit.

I would recommend the study of organs taken from animals affected with anthrax, and stained in the manner described, to those who, in spite of all the proof already furnished, do not yet regard it as a parasitic disease. The simple fact that death occurs in twenty-four hours after inoculation with the smallest drop of anthracic blood, provided that it contains bacilli or their spores, and that then almost all the capillaries

of the lungs, liver, kidneys, spleen, intestine, stomach, &c. (placed in absolute alcohol immediately after death), are found to be filled with enormous numbers of the same bacilli, has so evidently only one interpretation that no commentary is required. The investigator who still considers the presence of these organisms as accidental, quite immaterial, or merely accessory, must, before he can attribute the death of the animal to some other unknown ferment, consider likewise as immaterial the loss of the constituents of the blood which go to build up these innumerable bacilli, the accumulation of waste products which such a rapid interchange of material as the growth of the bacilli must of necessity involve, and also the disturbances in the circulation and in the nourishment of important organs induced by the plugging of most of the capillaries. But in that case there would be no reason why, in the case of trichinosis, scabies, and other parasitic diseases communicated by direct contact, some specific ferments in addition to trichinæ, acari, &c., should not also be supposed to be present.

CONCLUSIONS.

I AM well aware that the investigations above described are very imperfect. It was necessary, in order to have time for those parts of the investigation which seemed the most important and essential, to omit the examination of many organs, such as the brain, heart, retina, &c., which ought not to pass unnoticed in researches on infective diseases. For the same reason no record was kept of the temperature, although this would undoubtedly have yielded most interesting results. I have intentionally refrained from entering into details of morbid anatomy, as only the etiology interested me, and as I did not feel qualified to undertake a study of the morbid anatomy of traumatic infective diseases. I must therefore leave this part of the investigation to those who are better able to undertake it.

Nevertheless I consider that the results of my researches are sufficiently definite to enable me to deduce from them some well-founded conclusions.

In this summary I shall, however, confine myself to the most obvious conclusions. It has indeed of late become too common to draw the most sweeping conclusions as to infective diseases in general from the most unimportant observations on bacteria. I shall not follow this custom, although the material at my command would furnish rich food for meditation. For the longer I study infective diseases the more am I convinced that generalisations of new facts are here a mistake, and that every individual infective disease or group of closely allied diseases must be investigated for itself.

As regards the artificial traumatic infective diseases observed by me, the conditions, which must be established before their parasitic nature can be proved, were completely fulfilled in the case of the first five, but only partially in that of the sixth. For the infection was produced by such small quantities of fluid (blood, serum, pus, &c.) that the result cannot be attributed to a merely chemical poison.

In the materials used for inoculation bacteria were without exception present, and in each disease a different and well-marked form of organism could be demonstrated.

At the same time, the bodies of those animals which died of the artificial traumatic infective diseases contained bacteria in such numbers that the symptoms and the death of the animals were sufficiently explained. Further, the bacteria found were identical with those which were present in the fluid used for inoculation, and a definite form of organisms corresponded in every instance to a distinct disease.

These artificial traumatic infective diseases bear the greatest resemblance to human traumatic infective diseases, both as regards their origin from putrid substances, their course, and the result of post-mortem examination. Further, in the first case, just as in the last, the parasitic organisms could be only imperfectly demonstrated by the earlier methods of investigation; not till an improved method of procedure was introduced was it possible to obtain complete proof that they were parasitic diseases. We are therefore justified in assuming that human traumatic infective diseases will in all probability be proved to be parasitic when investigated by these improved methods.

On the other hand, it follows from the fact that a definite pathogenic bacterium, *e.g.*, the septicæmic bacillus, cannot be inoculated on every variety of animal (a similar fact is also true with regard to the bacillus anthracis); that the septicæmia of mice, rabbits, and man are not under all circumstances produced by the same bacterial form. It is of course possible that one or other of the bacteric forms found in animals also play a part in such diseases in the human subject. That, however, must be specially demonstrated for each case; *a priori* one need only expect that bacteria are present; as regards form, size, and conditions of growth, they may be similar, but not always the same, even in what appear to be similar diseases in different animals.

Besides the pathogenic bacteria already found in animals, there are no doubt many others. My experiments refer only to those diseases which ended fatally. Even these are in all

probability not exhausted in the six forms mentioned. Further experiments on many different species of animals, with the most putrid substances and with every possible modification in the method of application, will doubtless bring to light a number of other infective diseases, which will lead to further conclusions regarding infective diseases and pathogenic bacteria.

But even in the small series of experiments which I was able to carry out, one fact was so prominent that I must regard it as constant, and, as it helps to remove most of the obstacles to the admission of the existence of a contagium vivum for traumatic infective diseases, I look on it as the most important result of my work. I refer to the differences which exist between pathogenic bacteria and to the constancy of their characters. A distinct bacteric form corresponds, as we have seen, to each disease, and this form always remains the same, however often the disease is transmitted from one animal to another. Further, when we succeed in reproducing the same disease *de novo* by the injection of putrid substances, only the same bacteric form occurs which was before found to be specific for that disease.

Further, the differences between these bacteria are as great as could be expected between particles which border on the invisible. With regard to these differences, I refer not only to the size and form of the bacteria, but also to the conditions of their growth, which can be best recognised by observing their situation and grouping. I therefore study not only the individual alone, but the whole group of bacteria, and would, for example, consider a micrococcus which in one species of animal occurred only in masses (*i. e.*, in a zooglæa form), as different from another which in the same variety of animal, under the same conditions of life, was only met with as isolated individuals. Attention must also be paid to the physiological effect, of which I scarcely know a more striking example than the case of the bacillus and the chain-like micrococcus growing together in the cellular tissue of the ear; the one passing into the blood and penetrating into the white blood corpuscles, the other spreading out slowly in the tissue

in its vicinity and destroying everything round about; or again, the case of the septicæmic and pyæmic micrococci of the rabbit in their different relations to the blood; or lastly, the bacilli extending only over the surface of the aural cartilage in the erysipetalous disease, as contrasted with the bacillus anthracis likewise inoculated on the rabbit's ear, but quickly passing into the blood.

As, however, there corresponds to each of the diseases investigated a form of bacterium distinctly characterised by its physiological action, by its conditions of growth, size, and form, which, however often the disease be transmitted from one animal to another, always remains the same and never passes over into other forms, *e.g.*, from the spherical to the rod-shaped, we must in the meantime regard these different forms of pathogenic bacteria as distinct and constant species.

This is, however, an assertion which will be much disputed by botanists, to whose special province this subject really belongs.

Amongst those botanists who have written against the subdivision of bacteria into species, is Nägeli, who says,* "I have for ten years examined thousands of different forms of bacteria, and I have not yet seen any absolute necessity for dividing them even into two distinct species."

Brefeld † also states that he can only admit the existence of specific forms justifying the formation of distinct species when the whole history of development has been traced by cultivation from spore to spore in the most diverse nutritive fluids.

Although Brefeld's demand is undoubtedly theoretically correct, it cannot be made a *sine quâ non* in every investigation on pathogenic bacteria. We should otherwise be compelled to cease our investigations into the etiology of infective diseases till botanists have succeeded in finding out the different species of bacteria by cultivation and development from spore to spore. It might then very easily happen that the endless

* Die Niederen Pilze. München, 1877, p, 20.

† Untersuchungen über die Spaltpilze. Sitzungsbericht der Gesellschaft Naturforschender Freunde in Berlin. 19th Feb., 1878.

trouble of pure cultivation would be expended on some form of bacterium which would finally turn out to be scarcely worthy of attention. In practice only the opposite method can work. In the first place certain peculiarities of a particular form of bacterium different from those of other forms, and in the second place its constancy, compel us to separate it from others less known and less interesting, and provisionally to regard it as a species. And now, to verify this provisional supposition, the cultivation from spore to spore may be undertaken. If this succeeds under conditions which shut out all sources of fallacy, and if it furnishes a result corresponding to that obtained by the previous observations, then the conclusions which were drawn from these observations and which led to its being ranked as a distinct species must be regarded as valid.

On this, which as it seems to me is the only correct practical method, I take my stand, and, till the cultivation of bacteria from spore to spore shows that I am wrong, I shall look on pathogenic bacteria as consisting of different species.

In order, however, to show that I do not stand alone in this view, I shall here mention the opinion of some botanists who have already come to a similar conclusion.

Cohn * states that, in spite of the fact that many dispute the necessity of separating bacteria into genera and species, he must nevertheless adhere to the method as yet followed by him, and separate bacteria of different form and fermenting power from each other, so long as complete proof of their identity is not given.

From his investigations on the effects of different temperatures and of dessication on the development of bacterium termo, Eidam † came to the conclusion that different forms of bacteria require different conditions of nutriment, and that they behave differently towards physical and chemical influences. He regards these facts as a further proof of the necessity of dividing organisms into distinct species.

I shall bring forward another reason to show the necessity

* Beiträge zur Biologie der Pflanzen. Bd. I., Heft. 3, p. 144.

† Beiträge zür Biologie der Pflanzen. Bd. I., Heft. 3, p. 223.

of looking on the pathogenic bacteria which I have described as distinct species. The greatest stress, in investigations on bacteria, is justly laid on the so-called pure cultivations, in which only one definite form of bacterium is present. This evidently arises from the view that if, in a series of cultivations, the same form of bacterium is always obtained, a special significance must attach to this form: it must indeed be accepted as a constant form, or, in a word, as a species. Can, then, a series of pure cultivations be carried out without admixture of other bacteria? It can in truth be done, but only under very limited conditions. Only such bacteria can be cultivated pure, with the aids at present at command, which can always be known to be pure, either by their size and easily recognisable form, as the bacillus anthracis, or by the production of a characteristic colouring matter, as the pigment bacteria. When, during a series of cultivations, a strange species of bacteria has by chance got in, as may occasionally happen under any circumstances, it will in these cases be at once observed, and the unsuccessful experiment will be thrown out of the series without the progress of the investigation being thereby necessarily interfered with.

But the case is quite different when attempts are made to carry out cultivations of very small bacteria, which, perhaps, cannot be distinguished at all without staining; how are we then to discover the occurrence of contamination? It is impossible to do so, and therefore all attempts at pure cultivation in apparatus, however skilfully planned and executed, must, as soon as small bacteria with but little characteristic appearances are dealt with, be considered as subject to unavoidable sources of fallacy, and in themselves inconclusive.

But nevertheless a pure cultivation is possible, even in the case of the bacteria which are smallest and most difficult to recognise. This, however, is not conducted in cultivation apparatus, but in the animal body. My experiments demonstrate this. In all the cases of a distinct disease, *e. g.*, of septicæmia of mice, only the small bacilli were present, and no other form of bacterium was ever found with it, unless

in the case where that causing the tissue gangrene was intentionally inoculated at the same time. In fact, there exists no better cultivation apparatus for pathogenic bacteria than the animal body itself. Only a very limited number of bacteria can grow in the body, and the penetration of organisms into it is so difficult that the uninjured living body may be regarded as completely isolated with respect to other forms of bacteria than those intentionally introduced. It is quite evident, from a careful consideration of the two diseases produced in mice—septicæmia and gangrene of the tissue—that I have succeeded in my experiments in obtaining a pure cultivation. In the putrefying blood, which was the cause of these two diseases, the most different forms of bacteria were present, and yet only two of these found in the living mouse the conditions necessary for their existence. All the others died, and these two alone, a small bacillus and a chain-like micrococcus, remained and grew. These could be transferred from one animal to another as often as was desired, without suffering any alteration in their characteristic form, in their specific physiological action and without any other variety of bacteria at any time appearing. And further, as I have demonstrated, it is quite in the power of the experimenter to separate these two forms of bacteria from each other. When the blood in which only the bacilli are present is used, these alone are transmitted, and thenceforth are obtained quite pure; while on the other hand, when a field-mouse is inoculated with both forms of bacteria, the bacilli disappear, and the micrococcus can be then cultivated pure. Doubtless an attempt to unite these two forms again in the same animal by inoculation would have been successful. In short, one has it completely in one's power to cultivate several varieties of bacteria together, to separate them from each other, and eventually to combine them again. Greater demands can hardly be made on a pure cultivation, and I must therefore regard the successive transmission of artificial infective diseases as the best and surest method of pure cultivation. And it can further claim the same power of demonstrating the existence of specific forms of bacteria, as must be conceded to any faultless cultivation experiments.

From the fact that the animal body is such an excellent apparatus for pure cultivation, and that, as we have seen, when the experiments are properly arranged and sufficient optical aids used, only one specific form of bacterium can be found in each distinct case of artificial traumatic infective disease, we may now further conclude that when, in examining a traumatic infective disease, several different varieties of bacteria are found, as *e.g.*, chains of small granules, rods, and long oscillating threads (such as were seen together by Coze and Feltz in the artificial septicæmia of rabbits, see p. 9), we have to do with either a combined infective disease,—that is, not a pure one,—or, what in the case cited is more probable, an inexact and inaccurate observation. When, therefore, several species of bacteria occur together in any morbid process, before definite conclusions are drawn as to the relations of the disease in question to the organisms, either proof must be furnished that they are all concerned in the morbid process, or an attempt must be made to isolate them and to obtain a true pure cultivation. Otherwise we cannot avoid the objection that the cultivation was not pure, and therefore not conclusive. I shall only briefly refer to a further necessary consequence of the admission of the existence of different species of pathogenic bacteria. The number of the species of these bacteria is limited; for, of the numerous diverse forms present in putrid fluids, one or but few can in the most favourable cases develop in the animal body. Those which disappear are, for that species of animal at least, not pathogenic bacteria. If, however, as follows from the foregoing, there exist hurtful and harmless bacteria, experiments performed on animals with the latter, *e.g.*, with bacterium termo, prove absolutely nothing for or against the behaviour of the former—the pathogenic—forms. But almost all the experiments of this nature have been carried out with the first mixture of different species of bacteria which came to hand without there being any certainty that pathogenic bacteria were in reality present in the mixture. It is therefore evident that none of these experiments can be regarded as furnishing evidence of

any value for or against the parasitic nature of infective diseases.

In all my experiments, not only have the form and size of the bacteria been constant, but the greatest uniformity in their actions on the animal organism has been observed, though no increase in virulence, as described by Coze and Feltz, Davaine, and others. This leads me to make some remarks on the supposed law of the increasing virulence of blood when transmitted through successive animals, discovered or confirmed by the investigators just mentioned.

The discovery of this law has, as is well known, been received with great enthusiasm, and it has excited no little interest owing to its intimate bearing on the doctrine of natural selection (Anpassung und Vererbung). Some investigators, who are in other things very exact, have allowed themselves to be blinded by the seductive theory that the insignificant action of a single putrefactive bacterium may, by continued natural selection in passing from animal to animal, be increased in virulence till it becomes deadly though a drop of the infective liquid be diluted a quadrillion times. They have founded thereon the most beautiful practical applications, not suspecting that the bacteria in question have never been with certainty demonstrated.

The original works of Coze and Feltz, as also that of Davaine, are not at my disposal for reference; and I cannot therefore enter into a complete criticism of them. So far, however, as I can gather from the references accessible to me, especially from the detailed notices in Virchow and Hirsch's 'Jahresbericht,' no complete proof that the virulence of septicæmic blood increases from generation to generation seems to have been furnished. Apparently blood more and more diluted was injected, and astonishment was felt when this always acted, the effect being then ascribed to its increasing virulence. But controlling experiments to ascertain whether the septicæmic blood were not already as virulent in the second and third generations as in the twenty-fifth, do not seem to have been made. My experiments so far support and are in accordance with those of Coze, Feltz, and Davaine

in that for the first infection of an animal relatively large quantities of putrid fluids are necessary; but in the second generation, or at the latest in the third, the full virulence was attained, and afterwards remained constant.

Of my artificial infective diseases the septicæmia of the mouse has the greatest correspondence with the artificial septicæmia described by Davaine. If we were to experiment with this disease in the same manner as Davaine experimented, we would, if no controlling experiments were employed, find the same increase in virulence of the disease. It would only be necessary to use blood in slowly decreasing quantities in order to obtain in this way any progressive increase of the virulence that might be desired. I, however, took from the second or third animal the smallest possible quantity of material for inoculation, and thus arrived more quickly at the greatest degree of virulence. Till, therefore, I am assured that, in the septicæmia observed by Davaine, such controlling experiments were made, I can only look on an increase in virulence as holding good for the earlier generations. In order to explain this we do not, however, require to have recourse to the magical wand of natural selection; a feasible explanation can be very naturally furnished. Let us take again the septicæmia of mice, as being the most suitable example.

If two drops of putrefying blood be injected into such an animal there is introduced not only a number of totally distinct species of bacteria, but also a certain amount of dissolved putrid poison (sepsin), not sufficient to produce a fatal effect, but yet certainly not without influence on the health of the animal. Different factors must therefore be considered as affecting the health of the animal. On the one hand there is the dissolved poison, on the other the different species of bacteria, of which, however, perhaps only two, as in the example before us, can multiply in the body of the mouse and there exert a continuous noxious influence. Only one of these two species can penetrate into the blood, and if the blood alone be used for further inoculations, only this one variety will come victorious out of the battle for existence. The further development of the experiment depends entirely on the quantity

of the putrid poison, and on the relation of the two forms of bacteria to each other in point of numbers. If one injects a large amount of the septic poison and a large number of that variety of bacteria which increases locally (in this case the chain-like micrococci causing the gangrene of the tissue), but only a very small number of the bacteria which pass into the blood (here the bacilli), the first animal experimented on will die, as a result of the preponderating influence of the first two factors before many bacilli can have got into the blood and multiplied there. Of the blood of this first animal, containing, as it does proportionately very few bacilli, one-fifth to one-tenth of a drop must be inoculated in order to convey the disease with certainty. In the second animal, however, only the bacilli are introduced, and these develop undisturbed in the blood. For the infection of the third animal the smallest quantity of this blood which can produce an effect is then sufficient, and after this third generation the virulence of the blood remains uniform.

We may also imagine another case in which the increase of the virulence may go on through more than two generations without any modification resulting from natural selection and transmission from animal to animal. This would take place if several species of bacteria capable of passing into the blood were introduced into the animal at the first injection. Let us suppose, for example, that in the same putrefying blood which served for the foregoing experiment, the bacilli of anthrax were also present, there would be then contained in the blood of the first animal not only the septicæmic bacillus, but also bacillus anthracis, and of each only a small number; of the anthrax bacillus there would be even fewer than of the other, because in mice they are deposited chiefly in the spleen, lungs, &c.; while in the blood of the heart they are, even in the most favourable cases, only sparsely distributed. On the other hand, the anthrax bacilli have this advantage, that, provided they be inoculated in considerable numbers, they kill even within twenty hours, while the septicæmic bacilli only destroy life after fifty hours. In the blood of the second animal, therefore, both species of bacilli would be present in larger numbers than in the first, although not yet so numerous

as if either organism had been inoculated singly. Hence a larger quantity of blood is necessary to ensure transmission to a third animal. Perhaps this might be the case even in the fourth generation, till finally one or other variety of bacillus would alone be present in the blood injected. Probably this would be the septicæmic bacillus.

In this way the experiments of Coze, Feltz, and Davaine may admit of simple explanation and be brought into harmony with my results.

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EXPLANATION OF THE PLATES.

All the drawings have been made as true to nature as possible by the use of the camera lucida and of Zeiss's $\frac{1}{12}$ inch oil immersion objective. An object micrometer was used to determine the magnifying power.

PLATE I.

Fig. 5.—Longitudinal section of the ear of a mouse. Progressive gangrene of the tissue:—

a. Normal cartilage and on each side normal tissue.
b. Line of demarcation, accumulation of nuclei.
c. Gangrenous portion of the ear devoid of nuclei.
d. Transverse section of a vessel full of micrococci. × 25.

Fig. 8. — Marginal zone of a cheesy abscess in a rabbit. Lateral portion:—

a. Cloud-like masses of zooglæa.
b. and *c.* Smaller.
d. Smallest micrococcus colonies.
e. Accumulation of nuclei in the neighbourhood of the zooglæa.
f. Broken-down nuclei.
g. Dead portion of zooglæa. × 700.

Fig. 11.—Capillary vessel from the mucous membrane of the small intestine of a septicæmic rabbit:—

a. Nuclei of the wall of the vessel.
b. Oval micrococci. × 700.

PLATE II.

Fig. 1.—Blood of a septicæmic mouse, dried on a cover glass, stained with methyl violet, and mounted in Canada balsam. Red blood corpuscles are seen, and among them are small bacilli. × 700.

Fig. 2.—White blood corpuscles from one of the veins of the diaphragm of a septicæmic mouse. All stages of transition are shown from blood corpuscles which contain but few bacilli to those which have become converted into masses of bacilli. × 700.

Fig. 4.—Blood of a mouse affected with anthrax. Red blood corpuscles and anthrax bacilli. The specimen was prepared in the same way as that shown in fig. 1. The points of junction of the bacilli are too strongly drawn. × 700.

Fig. 12.—Section of the ear of a rabbit parallel to the surface of the cartilage. The morbid process resembled erysipelas :—

a. Ball-like accumulation of bacilli.

b. Accumulation of nuclei above the layer of bacilli.

c. Nuclei of flat cells connected with the cartilage below the layer of bacilli.

d. Bacilli arranged parallel to each other. × 700.

Fig. 2

Fig. 1

Fig. 12

PLATE III.

Fig. 3.—Vein of the diaphragm of a septicæmic mouse:—

a. Nuclei of the vascular wall.

b. Septicæmic bacilli.

c. White blood corpuscles which have become transformed into masses of bacilli.

d. Capillaries opening into the vein. × 700.

Fig. 7.—Marginal zone of a cheesy abscess cavity in a rabbit. Lower surface:—

a. Accumulation of nuclei at the outer border of the abscess.

b. Zooglæa, consisting of very small micrococci (these have been drawn too large in parts, more especially in the interior of the zooglæa).

c. Zooglæa, partially dead.

d. Dead zooglæa.

e. Remnants of nuclei. × 700.

Fig 7

Fig 3.

PLATE IV.

Fig. 6.—A portion of the cartilage and adjacent tissue in the vicinity of *c*, fig. 5. Magnified 700 diameters:—

a. Necrotic cartilage cells.

b. Chain-like micrococci in masses.

c. The same isolated. × 700.

Fig. 13.—Villus of a rabbit. Anthrax. The bacilli alone stained. × 250.

Fig. [illegible]

Fig. 6

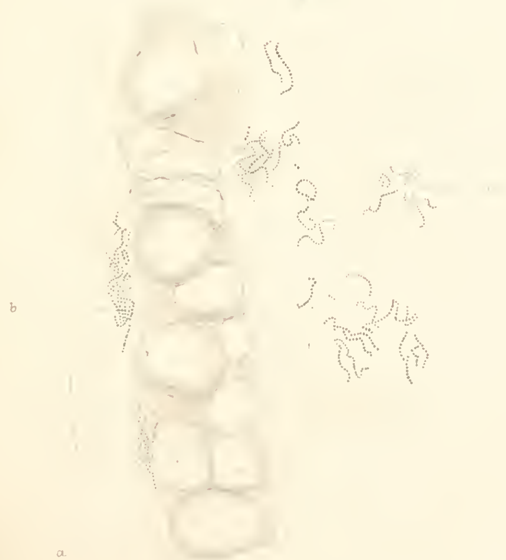


PLATE V.

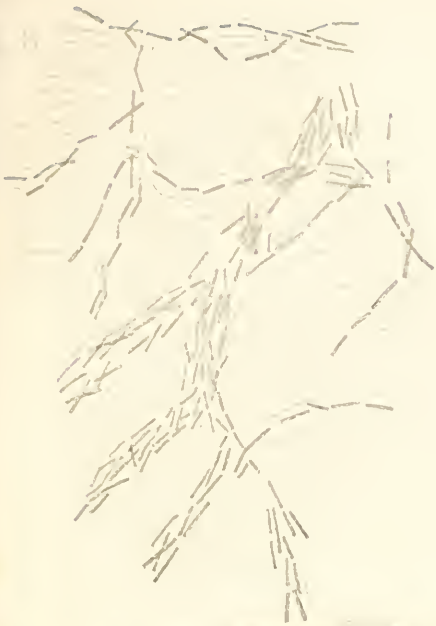
Fig. 9.—Vessel from the cortex of the kidney of a pyæmic rabbit:—

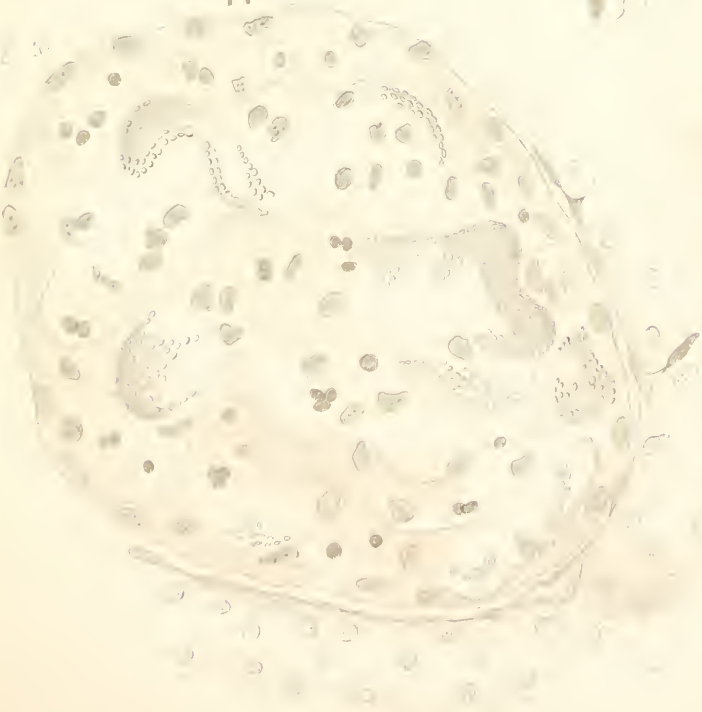
a. Nuclei of the vascular wall.
b. Small group of micrococci between blood corpuscles.
c. Dense masses of micrococci adherent to the wall and enclosing blood corpuscles.
d. Pairs of micrococci at the border of the large mass. × 700.

Fig. 10.—Glomerulus of a septicæmic rabbit:—

a. Capillary loop with oval micrococci spread out like a membrane.
b. Micrococci deposited on the walls of a capillary vessel.
c. Loop completely filled with micrococci.
d. Individual micrococci in a capillary vessel near a glomerulus. × 700.

Fig. 14.—A part of the vascular network of the same. × 700.





REPORT

PRESENTED TO THE

TWENTY-SECOND ANNUAL MEETING

OF THE

NEW SYDENHAM SOCIETY

HELD AT CAMBRIDGE,

AUGUST 8TH, 1880.

WITH

Classified List of Published Works

AND OTHER INFORMATION.

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Those marked with an Asterisk were not in office last year.

REPORT

PRESENTED TO THE TWENTY-SECOND ANNUAL MEETING OF THE NEW SYDENHAM SOCIETY.

THE series for the year 1879 consisted of the following works:—The second and concluding volume of Waring's Bibliotheca Therapeutica, a second part of the Society's Lexicon of Medical Terms, Guttmann's Manual of Physical Diagnosis, and a second Fasciculus of the Society's Atlas of Pathology. With the latter were included essays on the present state of knowledge as to the Pathology of the Kidney, by Dr. Greenfield, and as to that of the Spleen and Supra-Renals, by Dr. Goodhart. These papers were compiled at the request of the Council, and were freely illustrated by drawings from the microscope.

The series for the current year will probably comprise the following:—

I. A third fasciculus of the Society's Lexicon. (Already out).

II. The fifth and concluding volume of Hebra's Treatise on Skin Diseases, with index to the whole. (Already out).

III. Koch's Researches on the Etiology of diseases consequent on Wound Infection.

IV. A third Fasciculus of the Society's Atlas of Pathology, comprising diseases of the Liver.

V. A fourth Fasciculus of the Lexicon.

The Council has adopted, for reprinting, the classical Treatise of Dr. Stokes on Diseases of the Chest. This work, which has been always held in very high estimation by all authorities, has been for some time out of print. It will be edited for the Society, with short annotations, &c.,

by Dr. Hudson, of Dublin. It has also been decided to edit for the Society a selection from the works of Duchenne; and Dr. Vivian Poore has, at the Council's request, undertaken the preparation of the work.

The translation of Professor Charcot's Lectures on the Diseases of Old Age, and on certain Chronic Maladies, has been decided on. The work has been placed in the hands of Mr. William S. Tuke.

The preparation of the Society's Lexicon is, in the hands of its editors, Mr. Power and Dr. Sedgwick, progressing satisfactorily and as rapidly as the difficulties of the task permit. Three Fasciculi have been issued, and another is just ready. It is to be distinctly understood that the Fasciculi of this work are always issued as soon as ready. The Council is prepared to devote to it any portion of the year's income that may be requisite; and nothing but the onerous nature of the editors' task will be allowed to delay its publication.

The Balance Sheet has been audited, and is, as usual, appended.

BALANCE SHEET FOR 1879.

Receipts.

	£	s.	d.	£	s.	d.
Balance in hand, Dec., 1878 (see preceding Balance Sheet)				1082	13	1½
Subscriptions, 1 for 1862	1	1	0			
,, 1 ,, 1863	1	1	0			
,, 3 ,, 1864	3	3	0			
,, 1 ,, 1865	1	1	0			
,, 3 ,, 1866	3	3	0			
,, 2 ,, 1867	2	2	0			
,, 1 ,, 1868	1	1	0			
,, 2 ,, 1869	2	2	0			
,, 2 ,, 1870	2	2	0			
,, 3 ,, 1871	3	3	0			
,, 2 ,, 1872	2	2	0			
,, 2 ,, 1873	2	2	0			
,, 5 ,, 1874	5	5	0			
,, 9 ,, 1875	9	9	0			
,, 79 ,, 1876	82	19	0			
,, 136 ,, 1877	142	16	0			
,, 726 ,, 1878	762	6	0			
,, 1678 ,, 1879	1761	18	0			
,, 23 ,, 1880	24	3	0			
Volumes of Trousseau	128	12	0			
Fascic. of Atlas	12	12	0			
Other volumes	79	0	4			
	3033	3	4			
Less deductions per Local Secretaries	27	15	2			
				3005	8	2
				£4088	1	3½

Expenditure.

	£	s.	d.	£	s.	d.
Folio I. Artists, editors, and translators				692	3	6
,, II. Printers				838	2	8
,, III. Paper				408	0	0
,, IV. Bookbinders				339	13	10
,, V. Expenses of Management:—						
Agent's Salary and Percentage	264	14	7			
Secretary's Expenses	55	13	0			
Treasurer's Expenses	4	7	3			
Insurance	12	0	0			
Agent's Expenditure (chiefly carriage)	84	9	10			
Advertisements	21	14	10			
				442	19	6
Sum Total of Expenses				2720	19	6
Balance in hand, December 31st, 1879				1367	1	9¼
				£4088	1	3½

Examined, compared with the vouchers, and found correct, this 27th May, 1880.

Auditors { EDWARD CLAPTON,
F. M. CORNER,
SAMUEL FENWICK.

CLASSIFIED LIST

OF THE

SOCIETY'S PUBLICATIONS.

Medicine.

ON THE TEMPERATURE IN DISEASE: A MANUAL OF MEDICAL THERMOMETRY. By Dr. C. A. Wunderlich. (Leipzig). Translated by Dr. Bathurst Woodman. With forty Woodcuts and seven Lithographs.

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"In short, without pledging ourselves to Wunderlich as infallible, we may say, emphatically, that his is a masterwork, in which every part of his subject is considered with that thoroughness which comes of ripe knowledge and reflection. Let us add that Dr. Bathurst Woodman, following one or two laudable examples that have been set by other translators for the Sydenham Society, has enriched the work with notes of his own observations and those of other English writers, which are of no small value, and unquestionably do much to make the volume complete and full."—*Lancet*, April 20, 1872.

"The translator has rendered into readable English, and enriched with practical notes, a book which, even in its original form, has started into active work many physicians in England, France, and America, and which now, in its popular form, must render the diagnosis of disease infinitely more accurate."—*Medical Times and Gazette*, June 3, 1871.

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LECTURES ON CLINICAL MEDICINE, delivered at the Hotel Dieu, Paris. By Professor Trousseau. Five Volumes. Vol. 1, translated, with notes and appendices, by the late

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FIRST FASCICULUS.

Scrofula; Syphilis; and Lymph-Adenoma.—Plate I.

Fig. 1. Scrofulous Disease of the Kidney and Ureter. Fig. 2. Scrofulous Disease of the Kidney. Fig. 3. Scrofulous Disease of the Kidney. Fig. 4. A Mass of Syphilitic Deposit in the Cortical Substance of the Kidney. Fig. 5. Lymph-Adenoma of Kidney.

Nephritis after Diphtheria; Scarlet Fever; and Burns.—Plate II.

Fig. 1. Nephritis after Diphtheria.—Section of Kidney. Fig. 2. Subacute Nephritis after Scarlet Fever.—Outer surface of kidney. Fig. 3. Subacute Nephritis after Scarlet Fever. Fig. 4. Acute Nephritis after Scarlet Fever. Fig. 5. Subacute Nephritis after Scarlet Fever. Fig. 6. A cute Nephritis after a Burn.—Outer surface of the kidney of a child who died after a very extensive burn. Fig. 7. Acute Nephritis after a Burn.—Section of the same kidney.

The Granular Kidney in different stages.—Plate III.

Fig. 1. Extremely Granular Kidney. Fig. 2. Extremely Granular Kidney.—Section of the same kidney. Fig. 3. Less Granular (contracted) Kidney.—Outer surface of the right kidney taken from the same subject as the left kidney shown in Figs. 1 and 2. Fig. 4. Granular Kidney of Bright. Fig. 5. Contracted Granular Kidney, in section. Fig. 6. Contracted Granular Kidney; exterior. Fig. 7. Large Granular Kidney. Fig. 8. Large Granular Kidney with cysts.

Embolism; Infarction Processes from Pyæmia; Jaundice and Purpura; Scrofula.—Plate IV.

Fig. 1. Embolic Changes in Pyæmia. Fig. 2. Embolic Changes in Pyæmia. Fig. 3. Pyæmic Deposits in Kidney. Fig. 4. Pyæmic Deposits in the Kidney. Fig. 5. Results of Jaundice and Purpura. Fig. 6. A variety of the Scrofulous Kidney.—The substance of the kidney is wholly destroyed and replaced by cavities containing a white mortar-like substance.

SECOND FASCICULUS.

Diseases of the Kidney.—Plate V.

Fig. 1. Amyloid Disease of Kidney in advanced stage. Fig. 2. A section of the same Kidney. Fig. 3. The pale flabby Kidney. Fig. 4. The same organ seen in section. Fig. 5. Medullary Cancer of the Kidney.

Various Diseased Conditions of the Spleen.—Plate VI.

Fig. 1. Hodgkin's Disease of Spleen (Lympho-sarcoma). Fig. 2. Acute Splenic enlargement in Diphtheria. Fig. 3. Suppurating infarction of Spleen from a case of Ulcerative Endocarditis. Fig. 4. Embolic changes in Pyæmia. Fig. 5. Rupture of the Spleen.

Diseases of the Supra Renal Capsules and Spleen.—Plate VII.

Fig. 1. Cancer of the Supra Renal Capsule. Figs. 2, 3, 4. Adenoma of the Supra Renal Capsule. Fig. 6. Addison's Disease of the Supra Renal Capsule (in section). Fig. 5. Addison's Disease of the Supra Renal Capsule.—"Fibro-calcareous or strumous disease." Fig. 7. Tubercle of the Spleen (external surface). Fig. 8. Tubercle of the Spleen (in section). Fig. 9. Lardaceous Spleen.

Microscopic Pathology of Kidneys.—Plate VIII.

Fig. 1. Lardaceous Degeneration of the Kidney.—Section of cortex. Fig. 2. Lardaceous Degeneration.—*g*. A glomerulus from the same kidney, as in Fig. 1, which has undergone lardaceous degeneration and is becoming fatty. Fig. 3. Part of the same seen with a higher power, showing contents of one of the tubules. Fig. 4. Lardaceous Degeneration in earlier stage combined with interstitial fibrous change. Figs. 5 & 6. Lardaceous Degeneration (after Cornil). Fig. 5. Section showing the hyaline membranous wall of the tubules *a a* much swollen, stained violet-red, showing waxy degeneration. Fig. 6. Transverse section of one of the pyramids, near summit of cone. Fig. 7. Granular Contracted Kidney. Fig. 8. From the same.—A thickened arteriole surrounded by fibroid growth. Fig. 9. Partial Fibrous Degeneration of Malpighian body in slight chronic intertubular nephritis. Fig. 10. From the same kidney; showing early changes around Malpighian body. Fig. 11. Multiplication of Nuclei on glomerulus with adhesion of capillary tuft to wall of capsule. Fig. 12. Subacute Interstitial Nephritis with large white kidney. Fig. 13. Scarlatinal Nephritis. -Intertubular exudation in a case fatal on 7th day of fever. Fig. 14. Subacute Interstitial Nephritis. Fig. 15. Acute Catarrhal Nephritis, showing swelling and granular degeneration of epithelium. (100 diam.) Fig. 16. Part of the same seen with a higher power. Fig. 17. Section of cortex from a case of parenchymatous (catarrhal) nephritis at a later stage (so-called "fatty" kidney). Fig. 18. From nearly transverse section near base of pyramid in similar case. Fig. 19. Casts in tubes in interstitial nephritis (post scarlatinal). Fig. 20. Colloid cast, *b*, in tubule; *a*, unaltered epithelium.

Microscopic Pathology of the Kidney.—Plate IX.

Fig. 1. Scarlatinal Nephritis. Fig. 2. Shows two of the glomeruli from same section as Fig. 1. Fig. 3. Section from the same.—Part of the wall of a Malpighian body from which the capillary tuft has fallen out. Fig. 4. Scarlatinal Nephritis.—(From a case fatal about 12 weeks from attack of fever). Fig. 5. Scarlatinal Nephritis. —(From a case fatal 15 months after attack of scarlet fever). Fig. 6. From same kidney as Fig. 5, but in a deeper part of cortex, close to medulla. Similar growth of interstitial connective tissue. Fig. 7. Subacute Interstitial Nephritis, probably Scarlatinal, under low power; showing diffuse infiltration and cluster of dilated tubules. Fig. 8. Chronic Parenchymatous Nephritis (large white kidney) with little or no interstitial change. Section of cortex, showing changes in epithelium of convoluted tubules. Fig. 9. Kidney in leucocythæmia—to show localisation of changes around glomeruli and vessels. Fig. 10. Swelling of inner coat of small artery in granular contracted kidney. Fig. 11. Tuberculous Pyelonephritis. Fig. 12. Fatty Degeneration from Alcoholic Poisoning (after Lancereaux). Fig. 13. Fatty Degeneration in Cancer. Fig. 14. Individual epithelial cells from the preceding section; in various stages of fatty degeneration. Fig. 15. Cystic Degeneration of Kidney (after Lancereaux.) Fig. 16. From a cyst in kidney near base of pyramid. Fig. 17. Colloid Degeneration of Kidney. Figs. 18, 19, 20, and 21, illustrate the hyaline changes found in the splenic arteries in certain febrile conditions. Fig. 18. From a section through the spleen of a case of early scarlatina, showing hyaline degeneration of the coat of an artery, transversely cut. Fig. 19. Artery in longitudinal section. Fig. 20. Malpighian corpuscle from the spleen of a case of early scarlatina. Fig. 21. Part of the central and intermediate zone of the same Malpighian corpuscle as in Fig. 20, only more highly magnified (180 diam.) Fig. 22. Hodgkin's Disease. — Section of a spleen to show the overgrowth of the lymphatic sheath in Hodgkin's disease. (1 inch.) Fig. 23. Adenoma of the Supra Renal Capsule, showing the columns stuffed with fatty granules.

Microscopic Pathology of Spleen and Supra-renals.—Plate X.

Fig. 1. Capsulitis of the Spleen.—Vertical section of fibrous nodule in the capsule of the spleen, showing that the thickening of the capsule takes place by cellular growth in its deeper layers. Fig. 2. Fibrosis of the Spleen.—From the enlarged spleen of a ricketty child. Fig. 3. Fibrosis of the Spleen.—Showing a more advanced or fibrous condition spreading round some dilated veins. Fig. 4. Muscular Hypertrophy.—Over-growth of muscular trabeculæ in the spleen. Fig. 5. Muscular Hypertrophy. — Extreme stage of fibro-muscular growth in the spleen. Fig. 6. The Leucocythæmic Spleen.—Section of the edge of a Malpighian corpuscle, showing the compressed fibrous tissue between it and the splenic pulp. Fig. 7. The Leucocythæmic Spleen. — The pulp and stroma are normal. Fig. 8. Hodgkin's Disease.—The texture of a lymphoid noduie in the spleen of Hodgkin's disease. Fig. 9. Tubercular Spleen. (37 diam.) Fig. 10. Tubercular Spleen. Fig. 11. Induration and Atrophy. — A section of the spleen from a case of heart disease. Fig. 12. Lardaceous Spleen.—The sago spleen, showing the Malpighian corpuscles and small arteries mapped out by structureless hyaline lardaceous matter. Fig. 13. Lardaceous Spleen.—Transverse section of a Malpighian corpuscle, or small artery, with its surrounding lymphoid sheath. Fig. 14. Addison's Disease.—Vertical section of a supra renal capsule from the exterior inwards, to show the early changes in Morbus Addisonii. (250 diam.) Fig. 15. Addison's Disease.—Section of a supra renal capsule, to show the late, or fibro-calcareous, stage of Morbus Addisonii.

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Arranged according to the Year of Issue.

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1. DIDAY on Infantile Syphilis.
2. GOOCH on Diseases of Women.
3. MEMOIRS on Diphtheria.
4. VAN DER KOLK on the Spinal Cord, &c.
5. MONOGRAPHS (Kussmaul and Tenner, Graefe, Wagner, &c.)

1860. (*Second Year.*)

6. Dr. BRIGHT on Abdominal Tumours.
7. FRERICHS on Diseases of the Liver. Vol. I.
8. A YEARBOOK for 1859.
9. ATLAS of Portraits of Skin Diseases. (1st Fasciculus.)

1861. (*Third Year.*)

10. A YEARBOOK for 1860.
11. MONOGRAPHS (Czermak, Dusch, Radicke, &c.)
12. CASPER'S Forensic Medicine. Vol. I.
14. ATLAS of Portraits of Skin Diseases. (2nd Fasciculus.)

1862. (*Fourth Year.*)

13. FRERICHS on Diseases of the Liver. Vol. II.
15. A YEARBOOK for 1861.
16. CASPER'S Forensic Medicine. Vol. II.
17. ATLAS of Portraits of Skin Diseases (3rd Fasciculus.)

1863. (*Fifth Year.*)

18. KRAMER on Diseases of the Ear.
19. A YEARBOOK for 1862.
20. NEUBAUER and VOGEL on the Urine.

VOL. 1864. (*Sixth Year.*)

21. CASPER'S Forensic Medicine. Vol. III.
22. DONDERS on the Accommodation and Refraction of the Eye.
23. A YEARBOOK for 1863.
24. ATLAS of Portraits of Skin Diseases. (4th Fasciculus).

1865. (*Seventh Year.*)

25. A YEARBOOK for 1864.
26. CASPER'S Forensic Medicine. Vol. IV.
27. ATLAS of Portraits of Skin Diseases. (5th Fasciculus).

1866. (*Eighth Year.*)

28. BERNUTZ and GOUPIL on the Diseases of Women. Vol. I.
29. ATLAS of Portraits of Skin Diseases. (6th Fasciculus.)
30. HEBRA on Diseases of the Skin. Vol. I.
31. BERNUTZ and GOUPIL on Diseases of Women. Vol. II.

1867. (*Ninth Year.*)

32. BIENNIAL Retrospect of Medicine and Surgery.
33. GRIESINGER on Mental Pathology and Therapeutics.
34. ATLAS of Portraits of Skin Diseases. (7th Fasciculus).
35. TROUSSEAU'S Clinical Medicine. Vol. I.

1868. (*Tenth Year.*)

36. THE Collected Works of Dr. Addison.
37. HEBRA on Skin Diseases. Vol. II.
38. LANCEREAUX'S Treatise on Syphilis. Vol. I.
39. ATLAS of Portraits of Skin Diseases. (8th Fasciculus).
40. CATALOGUE of Atlas of Skin Diseases. (First Part.)

1869. (*Eleventh Year.*)

41. LANCEREAUX'S Treatise on Syphilis. Vol. II.
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1870. (*Twelfth Year.*)

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None of the Works published in 1876, 1877, 1878, or 1879 can be obtained separately unless by special arrangement. The series of each of these years can be had on subscribing for the year.

LAWS OF THE NEW SYDENHAM SOCIETY.

I.—The Society is instituted for the purpose of supplying certain acknowledged deficiences in the existing means of diffusing medical literature, and shall be called "The New Sydenham Society."

II.—The Society shall carry out its objects by a succession of publications, of which the following shall be the chief:—1. Translations of Foreign Works, Papers, and Essays of merit, to be reproduced as early as practicable after their original issue. 2. British Works, Papers, Lectures, &c., which, whilst of great value, have become from any cause difficult to be obtained, excluding those of living authors. 3. Annual Volumes consisting of Reports in Abstract of the progress of the different branches of Medical and Surgical Science during the year. 4. Dictionaries of Medical Bibliography and Biography. Those included under Nos. 1 and 2 shall be held to have the first claim on the attention of the Society; and the carrying out of those under Nos. 3 and 4 shall be considered dependent upon the amount of funds which may be placed at its disposal.

III.—The Subscription constituting a Member shall be One Guinea, to be paid *in advance* on the 1st of January annually, and it shall entitle the subscriber to a copy of every work published for that year. *No books shall be issued to any Member until his subscription for the year has been paid.*

IV.—The Officers of the Society shall be elected from the Members, and shall consist of a President, sixteen Vice-Presidents, a Treasurer, a Secretary, and a Council of thirty-two, in whom the power of framing Bye-laws and of directing the affairs of the Society shall be vested. Twelve of the Council shall be provincial residents.

V.—Five Members of the Council shall form a quorum.

VI.—The Officers of the Society shall be elected by ballot at the General Anniversary Meeting of the Society. Balloting lists of Officers proposed by the Council, with blank places for such alterations as any Member may wish to make, shall be laid on the Society's table for the use of Members.

VII.—The President, Vice-Presidents, and Council, shall be eligible for re-election, except that of the Vice-Presidents four, and of the Council eight, shall retire every year.

VIII.—The Council shall appoint local Honorary Secretaries wherever they shall see fit.

IX.—The business of the President shall be to preside at the Annual and Extraordinary Meetings of the Society; in his absence one of the Vice-Presidents, or the Treasurer, or any Member of the Council chosen by the Members present, shall take the Chair.

X.—The Treasurer, or some person appointed by him, shall receive all moneys due to the Society.

XI.—The money in the hands of the Treasurer, which shall not be immediately required for the uses of the Society, shall be vested in such speedily available securities as shall be approved by the Council.

XII.—The Council shall select the Works to be published by the Society, and shall make all arrangements, pecuniary or otherwise, in regard to their publication. In the event of any Member of the Council being appointed to edit any Work for the Society, for which he is to receive pecuniary remuneration, he shall immediately cease to be a Member of the Council, and shall not be eligible for re-election till after the publication of the Work.

XIII.—The Council shall lay before the Members at each Anniversary Meeting a Report of their proceedings during the past year, and also an account of the Receipts and Expenditure of the Society; and shall further cause to be printed and circulated among the Members an abstract of such Report and Accounts immediately after such Anniversary Meeting.

XIV.—The annual Accounts of the Receipts and Expenditure of the Society shall be audited by a Committee of three Members, selected at the preceding Anniversary Meeting from among the Members at large.

XV.—The Secretary shall have the management of the general Correspondence of the Society, and of such other business as may arise in carrying out its objects.

XVI.—The local Secretaries shall further the objects of the Society in their respective districts, and shall be in communication with the metropolitan Secretary.

XVII.—The Anniversary Meeting shall be held in the same town as, and at the time of, the Annual Meeting of the British Medical Association, notice of it having been given to all Members at least a week before the day fixed on.

XVIII.—The Members generally shall be invited and encouraged to propose Works, &c., and to make any suggestions to the Council they may think likely to be useful.

XIX.—The Works of the Society shall be printed for the Members only.

XX.—No alteration in the Laws of the Society shall be made, except at a General Meeting. Notice of the alteration to be proposed must also have been laid before the Council at least a month previously.

XXI.—The Council shall have power to call a General Meeting of the Members at any time, and shall also be required to do so within three weeks, upon receiving a requisition in writing to that effect from not less than twenty Members of the Society.

XXII.—All Special General Meetings of the Society shall be held at such place as the Council may appoint.

XXIII.—The Council shall meet at least once in two months, unless by special resolution to the contrary.

GENERAL INFORMATION.

A THIRD EDITION of the VOLUMES for 1859 was printed, and a Second of that for 1860. For subsequent years the First Edition was much larger; and it is not likely that any of the Volumes will be reprinted.

Most of the stones for Plates, &c., both those for the Atlas of Skin Diseases and those for printed Volumes, have been destroyed, and will not be reproduced.

A few complete Sets of the Society's Works are on hand, and can be obtained by new Members, but the number remaining is very small.

The Society is now in its Twenty-second year, and the cost of a complete Set to the end of 1879 is Twenty-one Guineas. Arrangements have been made by which new Members can obtain single Volumes, or sets of Volumes, from the Society's stock in hand. Some of the Volumes, of which a larger surplus exists than of others, can be purchased at fixed prices (for which see list.) The Society's Agent is empowered to make special arrangements with new Members who may wish to obtain any of the past Volumes.

CARRIAGE, &c.—The Society's Works are supplied free of cost to any address in London, Edinburgh, or Dublin; but the expenses of Carriage to all other places must be borne by the Members to whom they are sent. Members wishing to receive their Volumes by Book-post can do so by prepaying the postage. Members are requested to give detailed instructions respecting the mode by which they wish their Volumes to be forwarded, and also to remember that the Society's responsibility ceases when the Book has been delivered according to the instructions given. Members wishing to receive their Works by Book-post can do so by prepaying the sum of 2*s.* 6*d.* for the year.

The Subscription is One Guinea annually, to be paid *in advance.* The best mode of sending money is by Post-office Order, payable to Mr. HENRY KING LEWIS, at the London Office; or by Cheque to the

order of the Treasurer, Dr. Sedgwick Saunders. It is requested that in future all communications in reference to the payment of Subscriptions, or the issue of Books, may be made to Mr. Lewis, the Society's Agent, and not to the Secretary.

JONATHAN HUTCHINSON,
Hon. Secretary.

15, Cavendish Square, W.

*** Any Member wishing for additional Copies of this Report, &c., can obtain them by applying to Mr. Hutchinson; or to the Society's Agent, Mr. Lewis, 136, Gower Street, W.C. The Council will be much obliged by its distribution amongst those thought likely to join the Society.

PS.—The Society's Agent is prepared to supply, at fixed prices, CASES for binding the Lexicon, and PORTFOLIOS for the reception of the Plates of Skin Diseases.

NOTICE TO NEW SUBSCRIBERS AND LOCAL SECRETARIES.

New members who subscribe for not fewer than three past years at once will be allowed to select volumes from the surplus stock to the value of one guinea without additional payment (see page 29 for list of surplus stock and prices). The like privilege will be secured each year by any Local Secretary who has not fewer than ten members on his list whose subscriptions are paid before the end of March.

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And of Towns where it is desired that an appointment should be made.

The Council will be much obliged to any gentlemen willing to act as Local Secretaries in Towns where the appointment is vacant, if they will communicate with Mr. Hutchinson. *Any suggestions of suitable names will also confer a favour. The duties of Local Secretaries consist in arranging for the distribution of books, the collection of Subscriptions, and canvassing for new members.*

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